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Biology of human



The Biology of Human Starvation

"I suffered their summer famine with the nomads . . . Languor of hunger, the desert disease, was in all the tents. *Mâana lón*, 'we have nothing left,' said the people one to another. The days passed by days in this weakness of famine, in forgetfulness of the distant world, and the wasting life of the body. . . . Hither lies no way from the city of the world, a thousand years pass as one daylight; we are in the world and not in the world, where Nature brought forth man, an enigma to himself, and an evil spirit sowed in him the seeds of dissolution. And looking then upon that infinite spectacle, this life of the wasted flesh seemed to me ebbing, and the spirit to waver her eyas wings unto that divine obscurity."

CHARLES M. DOUGHTY, in *Travels in Arabia Deserta*, Chapter 17,
"The Moahib Summer Camp in Wady Thirba."

The Biology of
HUMAN STARVATION

by

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Psychology

Chapter 36. PSYCHOLOGICAL PROBLEMS IN STARVATION

Chapter 37. BEHAVIOR AND COMPLAINTS IN NATURAL STARVATION

Chapter 38. BEHAVIOR AND COMPLAINTS IN
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Chapter 39. INTELLECTIVE FUNCTIONS

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Chapter 42. PSYCHOLOGICAL EFFECTS —
INTERPRETATION AND SYNTHESIS

"Those who are hungry have no need of an elaborate philosophy to stimulate or excuse discontent . . . the good is enough to eat and the rest is talk."

BERTRAND RUSSELL, in *A History of Western Philosophy* (1945), Part II, Chapter 23, p. 747.

Psychological Problems in Starvation

IN ACCORD with the general historical development of the several branches of biology, psychology has been the last large area of scientific knowledge to be applied to problems of the effects of starvation. Although systematic, controlled studies on the physical aspects of semi-starvation were conspicuously rare, the large number of investigations made during World War II contributed significantly to our knowledge of the biochemistry, physiology, and pathology of prolonged, severe undernutrition. No comparable information exists on the psychological aspects of starvation and dietary rehabilitation. The few psychiatric studies on "natural" starvation that are available are based primarily upon unsystematic personal interviews. Standardized psychometric methods are of recent origin. The majority of tools used in the Minnesota Experiment for the study of personality and intelligence were developed only within the last two decades, and it should be recognized that their application under conditions of natural starvation would be very difficult.

Raw materials for the psychological study of starvation have not been lacking. Overt behavior is the human characteristic most immediately available for observation, and bodily sensations are subjectively the most impressive. Both show marked alterations in starvation. They are repeatedly mentioned in accounts of famine, from the earliest records until now, but for the most part in a casual and uncritical fashion. The welter of literary presentations of impressions, anecdotes, and personal rationalizations related to the psychology of starving persons is enough to produce despair as well as exhaustion in an attempt to winnow out the real and the significant. General accounts of famines, records of lost expeditions, and comments by relief workers constitute almost the whole descriptive material. This is the background against which the more precise and detailed data of the Minnesota Experiment must be projected.

At first thought the general frame of reference, the starting point, and the concomitant hopes and fears appear to be so different in the several circumstances of crop failure, physical mishap, prison brutality, and voluntary acceptance of an experiment that one would expect to find little in common among them. Obviously, differences in attendant conditions must modify the responses to the physical deterioration of starvation, but the bond between the physiological status of the organism and the "psyche" is closer than is sometimes realized. The dominance of the "body" becomes prominent under severe physical stress.

The *magnitude* of psychological changes resulting from semi-starvation may

be expected to exhibit marked variations, both in different individuals and in different conditions of starvation. The *direction* of change is a more fundamental characteristic and may be presumed to reveal qualitative aspects common to many conditions of caloric inadequacy, differing in a variety of details from the controlled and specific features of the Minnesota Experiment. In any case, it is easy to recognize many common features in the psychological changes induced by starvation in natural and experimental conditions. We have not been able to discern any substantial qualitative differences, and the counterparts of many particular responses in the Minnesota subjects are readily found in reports from the field.

Starved individuals are not rare in hospitals; the starved state is produced by a variety of disorders, and clinical material is readily at hand among patients with cancer, esophageal stricture, and anorexia nervosa. One might think, therefore, that useful psychological details from patients, obtained with quantitative psychometric methods and by trained psychiatrists, would be abundantly available. This is not the case, however. The psychology of cachexia has not been an active field of inquiry. There is a large literature on anorexia nervosa, but it is scarcely suitable for the present purposes to examine the psychological effects of starvation that is psychogenic in its origin. Cases of esophageal stricture caused by abortive attempts at suicide are likewise unsatisfactory. There is little help to be had for the present inquiry from ordinary clinical literature.

Before embarking on the systematic presentation of the psychological data from all sources in the succeeding group of chapters, it is desirable to consider the nature of the psychological problems associated with starvation and, specifically, the objectives of the Minnesota Experiment. A basis for the delimitation between physiology and psychology is needed; the field must be defined at the outset. The place of evidence from animal experiments requires comment. Both field reports and experiments on man have their limitations, theoretically and in terms of data thus far secured. Since much emphasis naturally falls on the Minnesota Experiment, it is desirable to consider the methods used in it. These are the aims of the present chapter.

Some of the general problems in the study of the psychological effects of semi-starvation are also encountered by the biochemist and the physiologist and have been remarked upon in previous chapters. The relevance of animal experiments, the problem of total versus partial starvation, and the effects of specific deficiencies, infections, and traumata as factors complicating the picture of caloric insufficiency are examples of considerations common to investigations into any of the facets of the biology of the starving man. The comments in this chapter are restricted to those points which especially pertain to the study of general behavior, intelligence, and personality in relatively prolonged human undernutrition.

Objectives of the Minnesota Experiment

The objectives of the Minnesota Experiment in the psychological area of the inquiry were essentially the same as in the morphological, biochemical, and physiological sectors, namely, to describe and analyze changes induced by al-

tered dietary intake, both during starvation and in the process of refeeding. Ideally, one would like to present a series of mathematical relationships between the dietary factor (or factors) and the various facets of the biology of the human organism. Unfortunately, many important aspects of behavior can be described at present only in qualitative or semi-quantitative terms. This limits the possibilities of establishing functional (in the mathematical sense) relationships between psychological changes and the degree of caloric deficit or the caloric level of refeeding, but it does not affect the general validity of the approach.

In a few variables, such as performance in intellectual tests, there was no important relationship to loss of body weight, within the range of semi-starvation obtained in the Minnesota Experiment. In some characteristics a simple linear relationship was indicated; for example, the changes in the Depression score during the period of nutritional rehabilitation were substantially in direct proportion to the level of caloric supplementation. The mathematical simplicity of such a parallelism should not create an illusion of simple biological relationships. Our understanding of the *intervening* variables, and therefore our capacity to explain the process of recovery in this or any other psychological characteristic, is severely restricted.

Explanation, in the sense of an analysis of biological phenomena into their constituent components, is obviously an undertaking fraught with hazards. This is true of such apparently simple findings as the reduction of basal metabolism; the difficulties increase at an accelerated rate as we move from the biochemical through the physiological to the psychological level. Consequently, the dominant concern in the Minnesota Experiment was with description rather than with explanation.

At times the temptation to speculate about the mechanisms of behavior was not resisted. This seemed reasonably safe when dealing with behavior closely related to the physiological alterations, such as the use of additional clothing to compensate for the lowered heat production of the body. But the more personality factors, in the narrower sense, enter into the picture, the more uncertain the ground becomes.

In general terms, it will be readily agreed that actual behavior may be considered a result of basic personality (including affective, conative, intellectual, motor, and perceptive facets), past experience, the status of the organism at a given time, and the stimuli provided by the particular circumstances of the moment. "Explanation" of a specific item of behavior involves two things: (1) more information, especially concerning factors determining the "historical reaction basis" (Driesch) of an organism, and (2) a theory regarding the mechanisms of behavior. Present-day theories of the dynamics of behavior are anything but safe and unequivocal guides for interpretation (or prediction). The question of what is relevant and irrelevant in the subject's past can be evaluated on the theoretical level in terms of probability, using the techniques of correlation analysis. In this way the relevance of such objective data as the birth order or the amount of schooling in withstanding a stress could be established. Many

events which may exercise a lasting influence on the behavior are truly individual occurrences and can be discovered and evaluated only clinically.

However, the difficulties of analysis are already present when we deal with simple psychophysiological data. The various sensations and complaints, such as hunger or weakness, may have a variety of origins; indeed, it is certain that the bases of complaints are complex and that the texture of the underlying factors is not identical from subject to subject or from time to time within the same subject. For example, in the case of "hunger" complaints we may be dealing with unusually strong unpleasant afferent discharges from the stomach, with alterations in the functioning (or sensitivity) of the nervous centers related to local physicochemical changes and general metabolic activity, or with a change in the direction of attention, indicating a "hypochondriacal" concern with bodily sensations which would have been unnoticed by the subject at times when his interests were directed toward other things than the "food complex." Questions of performance, which represents a product of capacity and effort, are in no way simpler than questions of sensation; the analysis into components is difficult and frequently impossible.

All attempts at explanation based on components have to meet the old problem of the whole and its parts. In the case of complaints and behavior the parts are not all known, and even those which are recognized are imperfectly defined. Questions of "explanation" might be considered as abstruse philosophical problems, the analysis of which leads quickly to borderlands of verifiable knowledge. Unfortunately, they are inherent in the process of generalizing the results obtained in the Minnesota Experiment. It should be noted that deterioration, or at least decrement, of spontaneous activity was observed in areas of behavior, such as the intellectual pursuits, in which no important alteration in capacity was demonstrated. This may be accounted for only in part by the fact that the laboratory tests of performance are as a rule short and that laboratory testing of endurance presents nearly unsolvable problems, especially when dealing with a large number of subjects. The primary fact is that motivation, the degree of "effort," may change independently of capacity. Consequently, from the theoretical point of view, it is not surprising that behavior in non-test situations does not always conform to the results of tests of performance capacity. In practical terms, this fact restricts severely the possibility of predicting the work which a semi-starved individual may be expected to accomplish under conditions of ordinary life.

Some aspects of semi-starvation directly relevant to the study of behavior have been discussed in previous chapters. The special senses were covered in Chapter 32. The neurological sensory characteristics, neuromuscular functions, and motor performance, including endurance in hard physical work, have been considered in Chapters 33 and 34. In the chapters to follow, after a survey of the psychological features of natural starvation, we shall discuss behavior and complaints with reference to the Minnesota Experiment and shall present the effects of semi-starvation on personality and intelligence. The discussion of general trends is supplemented by detailed case studies of both "normal" and "abnormal" reactions to the stress of semi starvation and slow rehabilitation. In the

concluding chapter on psychology an attempt is made to arrive at a synthesis of the facts and an interpretation of their broader significance.

Psychology and Physiology

Historically, experimental psychology has its roots in physics and physiology (Piéron, 1948). Some authors even at the present time consider psychology as a chapter — an important chapter — of physiology (Roger, 1946, p. 13). Others emphasize with vehemence the essential independence of psychology. This is not only true of some philosophical psychologists who are afraid that by a too close contact with physiology, psychology will degenerate into a “psychology without soul”; similar views are also expressed by experimental psychologists. For example, Skinner (1947, p. 31) is wary of physiological theories of behavior. Although a reduction of the facts and principles of psychology to physiology and to the still more fundamental disciplines of chemistry and physics is held possible, realization of even the first steps is considered to be a long way off. Skinner objects to the use of hypothetical neural processes in the construction of a theory of behavior and points out that “Valid neurological explanations of important psychological laws are not arrived at with a very rewarding frequency.” Skinner’s arguments are valid even though the front on which he attacks the problem is somewhat narrow (cf. Kantor, 1947).

The question of the interrelationship of scientific disciplines is by no means limited to a theoretical, completely disinterested level. We have pointed out elsewhere (Brožek and Keys, 1945a) that in the earlier days of psychology there were both scientific and social (and economic) reasons for emphasizing the “independence” of the young science, the fight for independence being waged against physiology on one side and philosophy on the other. From the scientific point of view there was a need for the development of new tools of observation and of a new conceptual framework; the pressure of tradition would have hampered this creative process, which required an atmosphere of full freedom. Socioeconomic factors entered in as well and were expressed, for example, in the reluctance on the part of the heads of the established departments to allow the youngster Psychology to grow like a weed while the parent Philosophy was losing territory, students, staff, and prestige.

By establishing the Laboratory of Physiological Hygiene on a frankly collaborative, interdisciplinary basis, the vexing problems of departmental prestige were eliminated, or at any rate markedly attenuated (Brožek and Keys, 1944). The focus of the investigations carried out at the Laboratory of Physiological Hygiene is “the man as a whole,” the functioning of the human organism in all its observable aspects. The criterion for the application of this or that method is pragmatic: Does or does not the specific method characterize a relevant aspect of the human organism?

It might be asked why the chapters on special senses and motor performance were included under the heading of physiology rather than psychology. We wish to point out, right at the start, that no definite classification of these functions as “physiological” or sharp delimitation of the area of physiology and psychology was implied. Responses in all the tests of sensory functions used here

were mediated by conscious experience and were reported by the subjects; consequently, these tests could be considered to measure "psychological" functions. The purist might speak of only those data as "physiological" in which the response to sensory stimulation is recorded "objectively" — that is, in terms of action potentials or of involuntary reactions of the organism. In this case, the chapter on special senses in any text on human physiology would become exceedingly slim. Actually, the amount of "introspection" in the tests of special senses is minimal, the subject acting essentially as a complex registration apparatus. This point was stressed in the instructions given to the subjects in all these tests; they were to assume a passive attitude and simply had the task of reporting when the flickering light fused into a steady light, and so forth. There was no "voluntary" component in the test performance in any sense which would make doubtful the admission of the data to the status of "natural science."

The scores on tests of motor performance are valid only when the subject exerts himself to the "maximum" (defined with reference to a given set of circumstances), and it is a part of the skill of the experimenter to select subjects and to structure the general experimental conditions and specific test situations in a way that is conducive to the maintenance of optimal motivation on the part of the subjects. The fact that in such tests we have obtained repeatable scores over a period of many months (see e.g. Brožek and Alexander, 1947, p. 164) indicates that the component of "effort" may remain at a relatively constant level for the periods of time demanded by nutritional research on man. Under such conditions the test scores may be regarded as indicators of the physiological fitness of the organism.

In the case of intellectual functions the situation is essentially similar. We were surprised again and again to discover how stable the intellectual test scores were in the face of severe environmental or metabolic stresses resulting in marked changes in the personality and the emotional status of the subjects (see e.g. Guetzkow and Brožek, 1947). This indicates, in functional terms, the high resistance of the central nervous system to deterioration, a fact that would be expected on the basis of physiological and biochemical considerations. The problems of measuring intellectual functions are more complex than in the case of motor performance, but the principles are essentially the same. Also, the isolation of the "primary factors" in the motor and intellectual areas of performance differs, if at all, only in degree.

We agree fully with Cobb (1944), who emphasized, for the benefit of neurophysiologists, that "much psychology is nothing but a more complex physiology, in short, cerebral physiology rather than the physiology of the nerve-muscle preparation" (p. 4). The real dividing line, although not entirely an unbridgeable gulf, is reached in the study of personality; this is fully recognized as psychological territory.

Limitations of Animal Experiments

An attempt to consider in an exhaustive manner the results of animal experiments on starvation would have disproportionately increased the bulk of the present monograph. Moreover, the relevance of the data from animal studies is

markedly reduced by the fact that they are largely concerned with acute starvation on the one hand or with specific dietary deficiencies on the other. Also, the problems studied in animal experiments are for the most part far from those of central importance in human semi-starvation. It is useful, however, to survey some of the types of questions related to hunger and starvation which have been approached by the techniques of animal psychology.

One such problem is the relationship between the diurnal variations of the hunger drive and the general (spontaneous, nondirected) activity of an animal, such as washing, scratching, and running. Using "activity cages" which permit registration of the movements of the animals, a rhythmic ebb and flow in the general motility has been observed by many investigators. The specific distribution of the periods of activity and quiescence depends on many factors, especially the frequency of feeding, but in all instances a rise in activity takes place near the feeding time. Richter (1927) used a tambour-mounted arrangement consisting of a large living cage and a small food cage, the motility of the two parts being recorded separately. He found that the entrance to the eating cage was preceded by a spurt of general activity in the living cage. The cycle was from 2 to 4 hours in length and appeared to be related to the periodicity of the hunger contractions of the stomach wall.

Shirley (1928) maintained rats under constant light and kept a constant feeding hour. The activity, recorded in terms of the number of revolutions of the drums in which the rats were kept, was greatest during the hour preceding feeding. Skinner (1936) obtained similar evidence by determining the rate at which the rats moved a lever which released pellets of food.

Skinner's approach illustrates the type of study on animal behavior in which hunger is used as an internal excitant for specific activity. Another type of specific activity used as an objective criterion of the hunger drive is crossing an electrically charged grid to get to a box containing food. In the apparatus constructed by Moss (1924), the crossing of the electrical plate depended on two factors: the strength of the *drive* (proportional, within limits, to the length of food deprivation) and the strength of the *resistance* (indicated by the size of the charge on the grid). In a typical experiment Moss kept white rats without food for periods varying from 12 to 144 hours. The electrical charge on the grid was relatively high (28 volts) and was kept constant; the strength of the hunger drive was then measured in terms of the percentage of the animals that crossed the plate after a given number of hours of fasting. These values were 0, 5, 20, 50, 80, 95, and 100 per cent for groups which had fasted for 12, 24, 36, 48, 72, 96, and 144 hours, respectively.

The technique was improved by animal psychologists working at Columbia University (Jenkins, Warner, and Warden, 1926) and was applied by them in a large series of experiments. In their arrangement the strength of the drive was expressed in terms of the number of crossings (and also of contacts and approaches to the plate) in an exposure period of 20 minutes. In an investigation by Warner (1928), in which white rats were deprived of food for a period up to 8 days, there was an initial increase in the number of crossings made within the standard test time, followed by a gradual decline. The maximum number of

crossings was the same for both males and females, but the peak of about 18 crossings was reached by the females after 2 days of starvation, by the males after 4 days.

Heron and Skinner (1937) studied the intensity of the hunger drive by recording the frequency with which the rats depressed a lever that released a food pellet once every 4 minutes; the technique was mentioned in connection with the diurnal variations of the hunger drive. The pressing of the lever is a learned response. The periodic release of a minute portion of food serves as a reinforcement designed to avoid extinction of the response. The frequency of the responses was studied daily for one hour, the time of the day being kept constant for each animal. The hunger drive, measured by the hourly rate of responses to the lever, followed in general a course similar to that charted by Warner (1928). The mean rate of the responses for the group of 13 rats increased rapidly during the first 24 hours of food deprivation, reaching a level of nearly 500 responses; it continued to rise in the subsequent 4 days, although more slowly than during the first day, and reached a maximum value of about 1100 responses on the fifth day. Then the number of responses began to decline and dropped to zero when the animals became too weak to move. The authors emphasized the large individual differences in the time course of the changes in the strength of the hunger drive, the maximum rates being reached by individual animals from the fourth to the thirteenth day of the starvation regimen. The typical individual curve showed a progressive rise throughout the greater part of the experimental period, followed by a precipitous fall in the terminal phase. Wald and Jackson (1944), who studied changes in the level of general activity (locomotion), observed a rise for a period up to 4 days. This fact fits in with the general results of the other studies in which more direct criteria of the intensity of hunger were used.

Siegel and Steinberg (1949) studied gross bodily activity in the conventional home cage, equipped with a photoelectric recording device, in relation to the duration of food deprivation. Under conditions of *ad libitum* feeding and watering, the mean activity level of 60 male albino rats was characterized by a half-hour score of 16.8. When the animals were divided into 4 matched groups and their activity was determined after 12, 24, 36, and 48 hours of food deprivation, the mean scores rose to 29.0, 36.1, 36.5, and 39.3, respectively.

In the majority of experiments on learning, hunger was used simply as one of the incentives to specific behavior (for summary and comparison with other incentives, see Munn, 1933, esp. pp. 318-19). Of some interest in the present context are studies, such as that made by Tolman, Honzik, and Robinson (1930), indicating that the task of running a maze is learned more rapidly by rats deprived of food for a longer period of time, provided they do not become actually debilitated. It is evident that we do not deal here with an increased "intellective capacity" but with an increase in motivation. The literature on food-seeking and food-selecting behavior was critically reviewed by P. T. Young (1941, 1948).

Some of the experiments on hunger are of relevance for the social psychologist. Thus Bird (1940, p. 35) pointed out that in addition to internal stimuli in

the form of muscular rhythms (hunger contractions of the stomach) and complex changes in body chemistry (which lead to eating behavior in the absence of the contractile tissue of the stomach), perceptual and social factors enter into the picture. For example, a hen will eat more from a larger pile of grain than from a smaller pile. Also, if a hen has finished eating it will start again if hungry hens gain access to the grain (see also Bayer, 1929).

As indicated earlier, the amount of research on the effects of semi-starvation as a physiological state rather than on "hunger" as an activity drive is small. In order to explore such aspects as physical performance capacity and endurance, a technique developed by Kniazuk and Molitor (1944) might be used. In their arrangement the animal's effort to avoid drowning provides maximal motivation.

The effect of severe semi-starvation on the functioning of the central nervous system, explored by the technique of conditioned reflexes, was studied by two of Pavlov's students, Rozental (1922) and Frolov (1922). The experimental work was done during the second half of 1918 and the early part of 1919 at a time when the food supply in Leningrad became grossly inadequate for both man and beast. In addition to incidental observations on a group of 13 dogs, the authors reported in detail on two animals that lost more than 50 per cent of their body weight and finally died. One of the earliest neurophysiological symptoms of starvation was the loss of differential inhibition — that is, loss of the capacity to restrict the conditioned response, originally associated with either of two stimuli, to only one of them (the "positive" stimulus). In the second stage, the response to well-established conditioned responses decreased in intensity; the reactivity of the central nervous system was lowered, and a state of somnolence set in. It became impossible to elaborate new conditioned responses. At the time when the responses to previously conditioned *artificial* stimuli, such as the sound of a bell, decreased and finally became completely absent, the responses to *natural* conditioned stimuli, such as the sight and odor of food, were still retained in considerable force. In the terminal phase even these responses were greatly reduced, while the unconditioned salivary reflex continued to function, although the response was also depressed.

In Frolov's starved dog (1922, p. 168) the drops of saliva during a standard period of collection varied from 17 to 26 when food was administered (the "normal" response is from 40 to 50 drops) and from 2 to 3 drops when the sight of food was used as the conditioned (natural) stimulus, the usual response to sight of food being 7 to 10 drops; this experiment was carried out a few days before death. Frolov pointed out that the sequence of the disappearance of the functions of the central nervous system under conditions of starvation (and other conditions of deterioration) is exactly opposite to the order in which the functions appear in the ontogenesis. The most recent and most complex types of response drop out first; the unconditioned reflexes last the longest time. More recent references as to the effect of hunger and diet on conditioned responses are given by Hilgard and Marquis (1940, p. 155).

Taken as a whole the animal experiments do not contribute significantly to the problems with which we were concerned in the Minnesota Experiment. For the most part they deal with hunger as a motivating factor rather than with the

effects of starvation on fitness and behavior, which was our focus. The work of Pavlov's school comes closest to the type of experimental work one would like to have for comparative purposes. Unfortunately that part of it which we were able to consult is fragmentary. As far as we know, no work has been done with animals on nutritional rehabilitation after semi-starvation.

Limitations of Observations on Behavior in "Natural" Semi-Starvation

Under the conditions of nonexperimental starvation and famine, the victims are affected by a number of other distressing factors, both physical and psychological, which tend to increase the stress but which cannot be separated, in their effects, from those resulting from semi-starvation alone. In famine, particularly modern war famine, the scarcity of food is frequently aggravated by inadequate fuel for cooking and for heating houses, by the lack of warm clothing, and by the disruption of the mode of living and occupational activities. Stavèl (1936, p. 140) argued that the psychology of people exposed to "natural starvation" is as much a psychology of fear and desperation as a psychology of hunger and food deprivation.

In World War II numerous opportunities for the study of the physical effects of undernutrition were present. Psychologically the conditions were complicated and little suited to the detailed study of pure relationships between food intake on the one side and behavior and emotional adjustment on the other. Frequently, as in Leningrad during the siege (Brožek, Wells, and Keys, 1946), the observations were made only on patients admitted to the hospitals or otherwise coming under special medical care; little or no information is available on the conditions in the population as a whole.

Sukhareva (1943, 1947), in her discussion of psychological disturbances in children during war, pointed out that they suffered not only from undernutrition but also from lack of sleep and from physical exhaustion. The children who lived in the German-occupied zones "lived in constant fear and anxiety, witnessing torture and executions of relatives and neighbors, and death of their loved ones. They themselves were often threatened, humiliated and beaten. Their homes were burned" (*ibid.*, 1947, p. 33).

Accessory factors which increase the effects of a reduction in food supplies were brought out well by Besançon (1945). In the unoccupied section of France the daily ration from 1941 on is reported to have varied from 1000 to 1500 Cal.; no actual data on the diets were given. For some groups, such as inmates of mental hospitals, the food shortage was particularly severe and starvation mortality was very high. No estimates of body weight losses in the population are given except for a mention that losses of 10 to 20 lbs. (4 to 9 kg.) were common and losses of 50 to 60 lbs. (22 to 27 kg.) were heard of. The deterioration resulting from inadequate food was accelerated by the added burden of muscular work. The transportation facilities became less and less adequate, and walking and bicycle riding replaced much of the train and bus travel. People had to stand for hours in front of shops and administrative offices. Some expended much physical energy in working their little gardens after regular work hours. Additional hardship was caused by the shortage of fuel. The people suffered

psychologically from the general effects of war. The threat of deportation and the actual deportations of friends and relatives were an important source of anxiety and worry. Contagious diseases, especially tuberculosis, were rampant. There was a shortage of soap and laundry services were poor, resulting in skin infections.

In the work camps for the prisoners of war in Japan (Curtin, 1946) the food was poor, inadequate in calories and deficient in proteins, fats, and vitamins; the daily caloric intake for the working men was estimated at 1500–2000 Cal. The suffering that resulted from the inadequate diet was seriously aggravated by the brutality of the prison life. At one stage in the history of the work camps all sick men were placed on half rations and the rare Red Cross parcels were given only to men who were able to work. The lack of adequate clothing was a serious handicap during the winter months and contributed to the deterioration of health. Lacking blankets, the men slept in pairs to keep warm. Their clothes wore into rags and they would wait to take the garments of the dying. Each man had only what he wore and in wet weather had to dry the clothes on his body. Shoes were a rare possession and the men wore wooden clogs or tied sacking around their feet. Bedbugs, lice, and fleas were an ever-present pest. Maltreatment was a continuously threatening possibility. Punishments were inflicted for little or no reason and included standing at attention for long periods, solitary confinement, and severe beatings, resulting frequently in black eyes, broken eardrums, lacerated scalps, broken ribs, and knocked out teeth. The work varied, but often it was unpleasant and injurious to health; for example, the men had to work on crushing machines in a brick factory and shovel coal, iron, and copper ore in the closed-in holds of ships, full of heavy dust. Medical attention was inadequate. The presence of sick and dying men in the camp could not but affect morale unfavorably.

Thus the prisoners of war were affected by the stresses of imprisonment in addition to the psychophysiological changes resulting from malnutrition (Wolf and Ripley, 1947). Cochrane (1946) coined the word *Gefangenitis* (*gefangen* = captured) to describe the particular psychology of prisoners taken by the Germans. In the transit camps the diet was inadequate, and it resulted in hunger pangs and irritability. But there were other factors conducive to irritability, such as overcrowding, deficient sanitation, and innumerable bedbugs. Nervous tension was kept at a high pitch by the guards' shooting inside the wire fences. In the permanent camps the British did not suffer from chronic hunger, owing to the Red Cross parcels, yet as time went on there seemed to be an increase in the frequency of attacks of bad temper and loss of initiative. The aggression tended to turn inward and to lead to neurosis. However, Cochrane comments, the majority of the neuroses had their roots in civilian life.

The situation was still worse in the Nazi camps for the civilian population. The horrors of existence even in the detention camps are difficult to grasp in full; the psychological atmosphere of the extermination camps is beyond our capacity for empathy. The official daily ration in the Auschwitz concentration camp was one liter of watery soup, 250 gm. of bread, and about 25 gm. of margarine or sausage or imitation honey, providing an estimated maximum intake

of 1000 Cal. per day (Adelsberger, 1946). This was a severe semi-starvation regimen, and the German camp doctors did not expect the inmates to survive much beyond 6 months, yet little information can be gained on the psychological effects of undernutrition as such. The camps were grossly overcrowded, with 500 to 1000 people herded together in blocks 100 feet by 20 feet. Sanitary facilities were utterly inadequate. There was a shortage of water. The inmates were infested with lice, and many had scabies. They were under constant threat of being subjected to the brutalities of the guards. But dominating all the thoughts and fears was the dread of death by gassing or burning. The extermination verdict was entered in the camp books under the euphemistic term of "transfer for special treatment."

In the matter of inflicting physical suffering, the Nazis achieved the highest distinction in the camps for Russian prisoners of war. Markowski (1945), a Polish physician who became a prisoner of war in 1939 and served as a surgeon in various camps and hospitals, reported that about 80 per cent of the Russians who were taken as prisoners of war in 1942 and 1943 died from underfeeding and exposure; in the camps located on German-occupied Polish territory the Russian prisoners of war were forced to live without a roof above their heads, with no huts or tents, sleeping in holes dug out in the fields, with no blankets even during severe winter.

Observations on the behavior of starving explorers are complicated by the severity of the climate, which increases the stress of inadequate food intake, and by the rigorous selection of the men who make up exploring parties. Only able-bodied individuals who are and wish to maintain the appearance of being "tough" are accepted. The point was brought out well by Greely (1886, Vol. II, p. 195) when he asked his readers "to bear in mind that the [diary] entries quoted were written by men patient in hardships, and always inclined to moderate, as a matter of pride, their great discomforts."

Methodological Limitations of Experiments on Man

Most of the experimental work on caloric undernutrition in both man and animals has been done under conditions of acute starvation. This is understandable in view of the far greater expense and difficulty of studies on semi-starvation. In the case of man it is a most serious problem to obtain suitable experimental subjects. Occasionally it is possible to find an individual who is willing to undergo a fast for some days or even weeks; candidates for months of slow starvation are normally just not to be had.

Some years ago there was so much public interest in fasting that several men became relatively professional in the "art" of going without food and were repeatedly the subjects of scientific study. These men, and a few other persons who were studied, were mostly extreme food faddists or otherwise far from being classifiable as psychologically normal. Such psychological results as were obtained from them are of dubious application to ordinary persons and populations. Succi, the experimental subject studied by several workers, was generally recognized as being psychologically abnormal. In one of his prolonged fasts, during which he was under medical observation, his scores in strength tests in

creased during fasting; this result may simply reflect his intense conviction about the benefits of fasting.

A major defect in most previous studies has been the lack of enough subjects to establish the generality of the results obtained. Single individuals have been used as noted above. In the study by Marsh (1916) there were only 2 subjects, the author and his wife. The experiment lasted 3 weeks, the intake being gradually reduced in the first week and gradually increased in the third week; no food was taken in the second week. Where the effects were opposite in the 2 subjects, as in memory tests (the man deteriorated and his wife improved), the results were comfortably explained as sex differences. In Marsh's experiment, also, the control (pre-starvation) data were inadequate. Glaze (1928) used 3 subjects, his wife, himself, and another man who previously had fasted for a total of 300 days in the belief that fasting is beneficial to health. The results of these experiments and criticism of them are presented in some detail in other chapters concerned with the functions studied by these authors.

A question which merits emphasis is that of pre-experiment standardization and training. In measuring the response of the human organism to a stress, such as exposure to heat or maintenance on a deficient diet, the measurements obtained during the successive stages of the stress are compared with the pre-experimental or control values. In tests in which the scores are influenced by practice during successive administrations of the test it is essential to bring the scores to a stable plateau before the start of the stress period. Performance in both manipulative (Franklin and Brožek, 1947) and intellective (Guetzkow and Brožek, 1947) tests shows marked improvement with practice, and if the experimenter wants to use such instruments he must provide ample time for pre-experiment practice.

The experimental work on starvation, both total (Benedict, 1915b) and partial (Benedict *et al.*, 1919), carried out at the Nutrition Laboratory of the Carnegie Institution in Washington, D.C., suffered greatly because adequate time was not given to pre-experiment training. Levanzin, who served as a subject in the total fast of 31 days, began to fast on the third day of his stay at the laboratory. It is known that in some tests used by Langfeld in that experiment, such as cancellation, performance is significantly affected by practice; the apparent improvement of performance on this test during the fast must be explained primarily (if not fully) on this basis. Under such conditions and in the absence of other adequate controls, no inferences can be made about the effect of fasting on mental capacity.

In the later experiment on chronic undernutrition (Benedict *et al.*, 1919), in which Miles carried out the psychological investigations and which is the only work that might have yielded information comparable to that of the Minnesota Experiment, it was impossible to secure an adequate base line for the experimental group in the psychological measurements. Only one testing session was held before the semi-starvation diet was started; this amount of practice was definitely inadequate to provide valid control values. The measurements obtained in the early part of the post-experimental period could not be used as a satisfactory equivalent of the pre-experimental base line because the men over-

ate and many suffered gastrointestinal upsets. Shortly after the end of the experiment most of the subjects left and were not available for further testing.

In the design of the Minnesota Experiment about 3 months were devoted to standardization, providing ample opportunity for pre-starvation practice and adjustment to the laboratory routine. Tests susceptible to the effects of practice were repeated at intervals of 3 to 4 weeks throughout the starvation and rehabilitation phases of the experiment; from our previous experience we knew that at this rate of repeated testing under normal conditions the performance would remain at a plateau. Consequently the observed deterioration of performance may be legitimately ascribed to the experimental regimen.

Certain limitations are inherent in laboratory work on man even when all methodological criteria are satisfied and the experiments are carried out over sufficiently long periods of time. Sorokin (1922, p. 91) stressed the point that laboratory starvation is carried out under the supervision of a competent staff, it may be ended at will, and there is no fear of death from hunger, no loss of energy in attempting to get food, and no pain of seeing the suffering of one's family. These conditions may be considered "limitations" only from the point of view of an exaggerated realism; for a scientific analysis there is a real necessity for separating experimentally the factors that operate in the uncontrolled situations. The Minnesota Experiment was concerned with the effects of diet, not of diet plus the varied factors that accompany famine. The ideal of investigating only one *independent* variable at a time is valid even though in biology the *concomitant* variables cannot be held as rigidly constant as in physics or chemistry. This inevitably results in a greater variability of the values of the *dependent* variables and imposes limits to generalization.

Methods Used for the Study of Behavior, Intelligence, and Personality in the Minnesota Experiment

A variety of techniques were used in the Minnesota Experiment for the study of the effects of semi-starvation on behavior, intelligence, and personality. The methods are described with reference to their technical aspects in the Appendix on methods. They ranged from quantitative performance tests to analysis of the qualitative, descriptive material contained in the diaries kept by the subjects. The diaries and clinical observations and impressions provide rich but diffuse sources of information. Descriptive generalizations about a group are always much less precise than the averaging of values for characteristics which can be estimated quantitatively. In the latter case the variability within the group may be economically described by such criteria as the standard deviation or the range of the scores. In verbal descriptions of behavior the grading of the intensity of a phenomenon is awkward, and the enumeration of the exceptions is cumbersome. At best, general impressions of a group somewhat resemble a photograph obtained by superimposing individual pictures of faces. The fundamental characteristics stand out but the fringes are fuzzy.

In dealing with quantitative data the individual features may be brought out by projecting the individual values against the general frame of reference provided by the means and the standard deviations obtained for the group. In order

to describe the individual differences in response to the semi-starvation regimen in full flavor and with a feeling of reality, the quantitative material is supplemented in selected cases by concrete biographical data and clinical observations. The purpose of the parallel use of a clinical approach and of standardized, quantitative techniques was to facilitate interpretation of the data and to provide for cross-validation of the findings.

No effort was spared in the attempt to cover all the important facets of the psychology of the semi-starved man. This does not mean that we are fully satisfied with the coverage or the techniques used. The difficulties involved in the investigation of psychological phenomena, and in their quantitative study in particular, are numerous, and the limitations of the present methods should be frankly recognized. The narrowness of the concepts of "intelligence" and "learning capacity," as defined with reference to the tests used to measure these characteristics, is obvious. The creative component of intelligence, the ability to adjust to new conditions, and the variety of the modes of learning are beyond the reach of our present instruments; so are initiative and endurance, without which no genuine intellectual achievement is possible. The personality area fares no better. Some of the personality inventories, such as those of Guilford and Martin, lack proper validation. The Minnesota Multiphasic Personality Inventory (MMPI) was derived within the framework of the current, and at times confused, nosology and was designed for clinical purposes; at the start of the experiment we were by no means certain that a repeated use of the MMPI would not affect its validity.

The "drive ratings" and the self-ratings in general suffer from severe methodological weaknesses. Attempts at a quantitative evaluation of bodily sensations are fraught with difficulties related both to the estimation of the range (from minimum or "normal" to maximum) of each item, such as hunger, and to the uncertainty of gradation of the intensity of the symptoms or complaints. Also, in long-term experiments in which the subjects have to compare their status at a given time of the experiment with their condition in the control period, the "normal" state can easily become intangible, receding beyond a point of clear visualization. The changes may be so slow as to be hardly perceptible, either to the subjects or to observers who associate with them all the time. This idea was well expressed by Dr. Richardson, physician to the expedition of John Franklin, when he described his impressions on rejoining Captain Franklin, from whom he was separated for a few weeks: ". . . no words can convey an idea of the filth and wretchedness that met our eyes on looking around. *Our own misery had stolen upon us by degrees, and we were accustomed to the contemplation of each other's emaciated figures*, but the ghastly countenances, dilated eye-balls, and sepulchral voices of Captain Franklin and those with him were more than we could at first bear" (J. Franklin, 1823, Everyman's Library ed., p. 406).

The critical comment on methods should not create an impression of methodological nihilism. There is much to be said in favor of modern psychometric methods. For example, the repeatability of scores obtained in intellectual tests under standard conditions is amazing (see Guetzkow and Brožek, 1947, esp. p. 359); the initial concern about the effects of repeating the MMPI was not borne

out and the inventory proved to be a very useful instrument. However, we wish to state clearly that the methods have definite limitations and imperfections. A critical appraisal of the general approach and of the specific techniques should help the reader in weighing the evidence properly and in evaluating the biological and social significance of the observations reported in the next six chapters.

Behavior and Complaints in Natural Starvation

BEHAVIOR is the expression of the interaction between environment, in the broadest sense, and the individual. In natural (i.e., non-experimental) starvation, adverse conditions in addition to the lack of food generally make up a large part of the total experience, and the resulting behavior has a complex basis. However, starvation or severe undernutrition is such an overwhelming experience that its more specific effects should be discernible even in relatively impure situations, complicated by physical environment, emotional trauma, disease, and physical exhaustion.

All accounts of famine and severe food shortage touch upon, and often describe at some length, the behavior of the victims. Observations on how people behave under these conditions are interspersed with comments on their complaints. Diaries and narratives of explorers and shipwrecked personnel tend to be rich in information on the subjective aspects of starvation, on bodily sensations and emotions. Reports from relief workers and travelers in areas of famine emphasize mass behavior and the organization – or development of disorganization – in larger units of society. Official documents from times of famine often contain items of inferential value – the frequency and type of criminal offenses, the passage of edicts and laws which reflect changed tendencies in the behavior of the population, and so forth. On these matters there is an enormous potential fund of information of all degrees of relevance, validity, and psychological detail, ranging from codes of law to novels. Only a few samples can be cited here; they are presented to allow such understanding and synthesis as may come from a succession of accounts of disaster and suffering in which the common factor of starvation is dominant. A considerable number of works of creative literature deal with hunger and starvation – Flaubert's *Salambo*, Leskov's *Udol*, Hamsun's *Hunger*, and so on. In some of these works there is a basis of thorough historical study or firsthand observations and personal experiences, but they cannot be examined here.

As a measure of economy the present discussion deals only with famine and starvation in relatively "civilized" (as opposed to primitive, illiterate) societies. This limitation, however, should not be accepted without recognizing the intrinsic interest of starvation and food shortages in primitive societies. The methods of procuring and preserving food, designed as they are to prevent starvation, are of interest from the point of view of social anthropology (cf. Lowie, 1938, pp. 288ff; see also Richards, 1932). In civilized societies hunger and starvation are exceptional conditions. In some primitive societies anxiety about food is a potent

and persistent cultural factor and finds direct expression in the basic personality structure of the members of a given society. Kardiner (1939, p. 219) described and analyzed a particular form of hypochondriasis in the Marquesan Islanders, tracing it to a chronic type of hunger anxiety.

The General Effects of Famine

Famine denotes the semi-starvation of many people – a substantial proportion of the population of some sizable area. If the food shortage is confined to a city or to a small area, the term *local famine* is appropriate. In real famine the onset is usually gradual, and the duration is measured in months or even years. The direct mortality (pure starvation death) may be small, but the indirect mortality is large when measured as the excess over the ordinary mortality of the region. Defined in this way, real famine has been a frequent visitor in the past and threatens to be with us, in some parts of the world at least, for many years to come.

Separate sections of this chapter portray behavioral manifestations as they have occurred in various episodes of natural starvation. A number of common features are apparent, some of which were, in fact, indicated in the earliest records, such as the description of famine in the Valley of the Nile some 6000 years ago (quoted in Chapter 1, p. 5). Another old account may be of interest here. Morgulis (1923, p. 15) quoted from a 13th-century Russian chronicle referring to famine in Novgorod: "A brother rose against his brother, a father had no pity for his son, mothers had no mercy for their daughters; one denied his neighbor a crumb of bread. There was no charity left among us, only sadness, gloom and mourning dwelt constantly within and without our habitations. It was a bitter sight, indeed, to watch the crying children begging in vain for bread, and falling dead like flies." Historical material on starvation and famine in the Middle Ages gives many such pictures; part of this was summarized, with numerous quotations from the original sources, by Curschmann (1900).

The psychological and social effects of famine were discussed in detail by Sorokin in his unfinished monograph (1922) and again in connection with his general treatise on *Man and Society in Calamity* (1942). Sorokin noted that the cognitive processes, beginning with perception, become directed more and more to objects and thoughts connected with the calamity. In the case of famine, food becomes the central topic of conversation and writing. It intrudes constantly into the consciousness of the starving and disturbs the association of images and the flow of ideas. Coherent and creative thinking is impaired. The amount of time and energy spent in seeking food increases until the major part of the waking hours are so engaged, directly or indirectly.

As all thought and activity turn toward food, other normal activities are repressed or eliminated. Sex activities are reduced or cease. Sorokin distinguishes 3 mechanisms effecting this: a decrease in the physiologically determined sex drive, apathy toward indirect sex activities (such as courtship), and a suppression of activities which are incompatible with food-seeking efforts (e.g. marriage and social gatherings). The increase in women offering themselves for prostitu-

tion is not a refutation of this generalization, because it is the result of a loss of moral restraints and a subordination of them to food seeking.

In both primitive and civilized societies affected by famine the social ties are loosened, the usual social amenities and graces are dropped, wives and children are abandoned, and homes are left. Together with a general disregard of property rights, there is a revaluation of all property, reflecting the enhanced demand for food and those things which may be bartered for food. In periods of famine there is an enormous increase in thievery; violent crime increases, too, but apparently to nothing like the same extent unless there is total chaos and lack of governing authority. Riot and rebellion are engendered by minor hunger and deprivation, but real starvation makes for relative tractability. Though moral and social standards may be lost, lethargy and weakness are powerful deterrents against strong action. Some of the characteristics which starvation induces in all men are rather permanent traits of peoples among whom famine is endemic.

A recurrent famine phenomenon is the mass movement of populations from areas of food shortage. In the Orient such movements converge on the cities, the villagers abandoning their unproductive farms. This is the direction of migration in areas, as in India, where in the best of times the farmer's produce is only a bit more than his direct food needs; in the cities there are larger food reserves and with luck he may get a share. In Europe, where the productivity of the individual farmer is vastly greater, the urban dweller goes to the country in time of scarcity. In both cases the more vigorous and unfettered persons outdistance their fellows and thereby fare better.

By the extension of food-seeking activities, including migration, famines produce a change in the social structure. This is more important when the migration is toward the cities; the rural migrants can only hope to work and eat in the cities and they tend to stay there, at least for the duration of the famine. The city dweller, however, cannot stay in the country; he makes forays into it to buy or barter for food which he carries back to the city.

It has been remarked that a constant product of semi-starvation is black depression. It might be thought that with this dominating frame of mind the continuance of life in the appalling conditions so characteristic of famine would be intolerable to many. Suicide does occur but not with any extreme frequency. Episodes like that in India in 1291 when whole families drowned themselves (Elliot, cited by Loveday, 1914, p. 18) are decidedly rare. Far more frequently observers have been surprised at the tenacity with which famine victims cling to life under hopeless, degrading, and painful conditions.

There are few acts so basically revolting as cannibalism. However, to eat the bodies of the dead may not seem an unreasonable last resort to save the living. Surprisingly, the practice is never very common. Among Orientals particularly, where vast numbers are so often reduced to the extremity of want and the imminence of starvation death is recognized from bitter experience, cases of cannibalism are so few as to excite wonder. Perhaps the explanation is in the tremendous power of religious teaching; in the Orient the concept of the complete dissociation of body and soul is less fixed than in the Western world.

Obviously general behavior and mass psychology in famine must be affected by previous experience. Mallory (1926) stated that in a period of 2019 years (from 108 B.C. to 1911 A.D.) there were recorded 1828 famines in China. The history of India is similar. Clearly it would be inadmissible to predict mass behavior in the Western world, where famines have become relatively rare in the past 150 years, on the basis of observations in India and China. Some features, or at least tendencies, determined by the general physiological effects on the psychology of the individual are common to all starving people. But the behavior of man in society is also affected by other factors, including the structure and experience of the society of which he is a part.

It has been observed that in India the fear of famine can itself create a famine. Anxiety to build food reserves (hoarding) drives up prices, intensifying the fear, and all those who have food stocks become reluctant to part with them. Moreover, poor crop prospects and some developing scarcity induces many to migrate in the hope of getting to an area of greater abundance before there is famine. Farms and villages are abandoned, and the migrants may end in a sorry state, as in the migration from Travancore in 1943 and 1944 (Sivaswamy *et al.*, 1945).

In every famine it must be clear to the victims that the food shortage can be expressed as the relationship between their total food supply and the number of persons to be fed; if there were fewer people there might be enough to go around. Recognition of this fact in China was expressed in the custom of selling the children, particularly the girls, and in infanticide. But such measures, sporadically applied as they must be, are not curative of the population pressure, and it might even seem that the procreative instinct is intensified by the recurrent threat of famine.

The psychological aspects of the more recent and better documented incidents of famine and starvation are discussed in some detail in subsequent sections of this chapter.

Semi-Starvation in London, 1837-1838

The observations made by Howard (1839) on complaints and behavior in severe undernutrition in London in the fall and winter of 1837-38, when unemployment was general, constitute an interesting document. There was no famine in the proper sense, but semi-starvation was widespread among the working classes.

"The first indications of a deficiency of food . . . are languor, exhaustion, and general debility, with a distressing feeling of faintness and sinking at the praecordia, chilliness, vertigo, and a tendency to syncope, unsteadiness of movements, the voice weak and tremulous. Mental powers exhibit a languor and dullness proportionate to the degree of physical debility. The sufferer is listless and depressed, and often manifests a remarkable apathy to his condition." In more progressed cases "The feeling of prostration becomes quite overpowering, and the exhaustion and muscular weakness so great, that the erect posture can, with difficulty, be maintained. The depression of all the vital and mental powers is fearfully augmented. . . . Dizziness, transient dimness of vision, staggering and

syncope are common and very apt to be produced by erect posture. . . . Notwithstanding general languor, however, the patient sometimes manifests a highly nervous state; he is startled by any sudden voice and worried by the most trifling occurrences" (*ibid.*, p. 27).

Indian Famine of 1876-1878

Of psychological interest is the description by Digby (1878) of the Indian famine of 1876-78, a disaster due to rain failure. Early in the fall of 1876 anxiety about the crops was reflected in a rise in food imports, especially rice, to Madras. Toward the end of October apprehension about the imminent danger of famine became widespread. The people were seized by panic. Prices increased by 100 and even 200 per cent. Quoting from a government document, Digby described the situation as follows: "The rise [in the prices of food] was so extraordinary and the available supply, as compared with the well-known requirements, apparently so scanty, that merchants and dealers, hopeful of enormous gains, appeared determined to hold their stocks for some indefinite time and not part with the article which was becoming of such unwonted value. . . . retail trade up-country was almost at a standstill. Either prices were asked which were simply beyond the means of the multitude to pay, or shops remained entirely closed. Grain riots by hungry mobs of men, women, and even children, and more serious dacoities [gang robberies] accompanied by violence, followed in many parts of the country. . . . These disorders, though speedily and effectually suppressed, added another element of confusion and danger."

The distress was felt in an area covering 80,000 miles and inhabited by some 20,000,000 people. In the country districts people left their homes, wandering over the countryside and migrating to large towns. Relief employment and relief feeding were instituted but were inadequate as compared with the need. Some districts surrounding Madras were especially hard hit by the famine. With little or no relief employment made available, the inhabitants flocked to the city. "Melancholy specimens of emaciated beings" is Digby's characterization of these unfortunate people, some of whom attempted to keep alive by picking up the grains that fell from rice carts. People were dying of starvation in the streets. Social bonds were strained, as reflected in the cruel treatment and abandonment of children by their parents.

In rural areas people attempted to subsist on roots, some of which were not fit for human consumption. An incident is recorded in which women worked for 6 to 8 hours to secure a few handfuls of grain from ant holes. Some individuals of low caste kept themselves alive by feeding on the carcasses of dead cattle and by eating snakes and lizards.

By January 1877 the number of deaths due to starvation and diseases resulting from malnutrition was estimated at 65,000 in the Madras Presidency (population 16 million) and 13,000 in the province of Mysore. By January 15, 1877, up to one third of the population was on relief in some districts. Prices rose to nearly five times the pre-famine level. In visiting one of the relief houses in March, Digby saw some 800 individuals in every stage and every variety of starvation

—men and women bloated with dropsy and others who resembled moving skeletons, listless children, all loose skin and bone.

The appearance of the child victims of starvation was portrayed vividly in the second volume of Digby's book, which deals with relief operations: "The head looking unnaturally large by contrast with the emaciated trunk, the shoulder blades projecting as if they had been inserted by mistake in too small a carcass, the arms and legs shrivelled to the size of their bones, except at the knees which are swollen. Then the expression of bleared little faces. The vacant fixed look in their eyes, the drawn cheeks and lips, and the premature air of resignation" (Digby, 1878, Vol. II, p. 141).

The system of death registration in the provinces became ineffective, and the mortality figures for the year 1877 are too low (42,498 in February 1876 vs. 94,545 in 1877, excess 52,047; 35,863 in March 1876 vs. 91,119 in 1877, excess 55,256).

Because of the inadequacy of the relief diet, the fitness of the men in emergency employment gangs deteriorated. A tax collector of a provincial district considered few of the men fit for hard physical work. "Many were fine men of the *ryot* class, who, not long ago, were strapping fine fellows, but who are now losing, as I ascertained feeling them, all hardness of muscle and roundness of form. The women were in equally bad condition, and several of the very bad were little more than animated skeletons." It was also stated that a number of persons had to be taken out of the work gang and put on straight relief because they were unable to do work.

In May the severity of the distress was still rising, as the private sources of charitable aid were largely exhausted; the scorching heat added to the suffering. The weak and emaciated were shifted at a faster rate from relief work to gratuitous relief. The death rate in May 1877 was 92,355 as compared with 39,557 in 1876, or an increase of 52,798. By the middle of June, hopes for the rainfall usually brought by the southwest monsoons began to sink. In a few districts in which the meteorological conditions would have allowed cultivation of the soil, the men were apathetic and without the means for farming. A government officer wrote: "In every village I visit I find the *ryots* in a condition of idle despair, because they have neither cattle, nor seed, nor fodder; they generally cannot even borrow money to purchase bullocks, as their lands have passed out of their possession, having been sold outright or mortgaged beyond redemption. . . . The thatch from most of the houses has already been consumed in the maintenance of the few cattle that are yet in existence."

In some relief camps the people were becoming unmanageable and demoralized. It was difficult to make them retain their places in the ranks while waiting for food; they tended to rush from one place to another if there seemed to be a chance that they would be served earlier. A visitor to one of the camps wrote: "I was very much struck with the grumbling, fault-finding, quarrelsome spirit of the people. Some were quiet enough; they evidently had no strength to grumble or fight. But others, hundreds of them, swarmed round the visitors and complained of almost every possible thing. The quantity was not enough, the

rice was not good enough, the addition to the rice was not tasty enough, the meals were not punctual enough. . . . Quarrels, too, were going on."

The famine increased criminality and the number of prisoners in the central jail in one of the district towns (Salem) more than doubled; many persons were said to have deliberately committed crimes in order to get board (and room!) at the expense of the government. It was a sad commentary on the social system that the diet of the criminals was incomparably better than the ration for the population on relief. The death rates in June and July 1877 were 95,770 and 134,433, indicating an increase of 53,739 and 86,118 above the 1876 figures.

The crisis was reaching its culminating point. In Madras camps containing about 3000 inmates some 30 persons were found dead morning after morning. Infants were found on the roads, deserted by their parents, and in Madras mothers were offering children for sale. There were reports of cases of cannibalism, but this was certainly not a common occurrence. Finally, in September, the rains came, preserving some of the crops and making possible a relatively rapid recovery of the country. People began to return to their homes and their work. The northeast monsoons, arriving in November, provided an ample water supply, and fodder became plentiful.

Russian Famine of 1918-1922

Valuable material, based on firsthand and systematic observations on the effects of the Russian famine following in the wake of World War I and the Revolution, was presented by Sorokin (1942). The description is spread over nearly the whole length of his monograph, because it is used for illustrating the various facets of the sociology of famine in conjunction with other great calamities. We shall attempt to bring together the main threads.

In the years 1918-22, the period of widespread shortages, food was an absorbing topic in individual conversations as well as in public speeches. The newspapers devoted a great deal of space to the questions of food. Sorokin cites a report by a physician in the department of public health in the city of Viatka to the effect that "food topics incessantly occupied the whole consciousness of the pre-school and school children. Conversation about meals was the only possible approach to them. To everything else they reacted either irritably or negatively." The same author noted in children a decrease of attention and a decline in mental work, together with a marked fatigability. Sorokin recorded the complaint of intellectual workers about a decline in mental capacity and energy. Work performance in jobs requiring concentration and accuracy, such as printing and bookkeeping, is said to have deteriorated.

The amount of time devoted to food-seeking activities rose dramatically, amounting up to 70-95 per cent of all the waking hours, even in relatively well-to-do social groups such as the university students at Petrograd. This includes preparation, cooking, and eating of foods as well as such auxiliary activities as getting ration cards and standing in bread lines. Food became the largest item in the budget. Frequently the total income was inadequate to cover the expenditure for food, and the starving city population sold or exchanged everything that would sell or could be bartered for food.

The symptoms of personal and social deterioration were numerous. There was an increase in the incidence of mental illness and of suicide among both the adults and the children. Prostitution, under the pressure of hunger, became common. Children in large numbers were abandoned by their parents. Cannibalism was practiced. Crimes against public and private property, such as theft and robbery, assumed fantastic proportions. As to the behavior of children, Sorokin quotes the following passage from Aronovitch's report: "[In both public homes for children and private homes] there was observed a pathological greed for food. Children exhibited violent irritation, anger and hatred. They would fight to obtain a warmer place near the stove, or a larger ration during the distribution of food . . . they stole food from one another and from the administration of the children's homes; the stronger snatched food from the weaker; they lied shamelessly for a chance to obtain food."

The general picture of famine in Russia in the years 1918-22 could be further documented by numerous other reports. Ivanovsky (1923) commented on the bent body, the uncertain gait, the skin that lost elasticity and became wrinkled, the general senile appearance, and the weakening or even total disappearance of the sexual impulse. Hassin (1924), describing conditions during the Russian famine of 1922 on the basis of firsthand information, noted severe emaciation with disappearance of subcutaneous fat, accumulation of edema fluid, exhaustion, apathy, indifference to personal appearance and cleanliness, somnolence, loss of sexual desire, resignation, and in the late stages the immobility of the starved. The over-all apathy was strangely combined with heightened irritability; Ogranovitch (1919) reported that women killed other women in quarrels over their place in a queue.

The bulletins of the American Relief Administration contain many revealing passages concerning the effects of the Russian famine. The average ration in Petrograd in 1921 was estimated as 1200 Cal. daily, with the food consisting chiefly of very black bread and soup made from dry vegetables. There was no fat in any form. Some 50 per cent of the children in Petrograd were without shoes and in rags. There was no woolen clothing, practically no shoe leather, no soap; there was an oppressive lack of fuel. Moscow presented a picture of disorganization. The people were listless, in shabby clothing, having as the primary object in life the getting of food and clothing (American Relief Administration Bulletin, Ser. 2, No. 1, November 1921, pp. 7-14).

The report on famine in the Kazan district (*ibid.*, No. 20, January 1922, pp. 6-9) contains the following concrete description of starvation: "[In Lubimovka] we saw an entire family that was actually dying of starvation. . . . When we entered, the father was lying on a wooden bench, and he raised himself slowly and with difficulty. He was a man of a huge frame, who had wasted away to a skeleton. His clothes hung on him in rags. . . . He was so weak that his talk was only a mutter. . . . The mother and two girls were lying in the cubby hole over the stove, Russian fashion, and occasionally moaning. The boy, who was about 15, with a fine face, and splendid, frank eyes, was so weak that he could not even smile, or show any animation whatever."

The American relief workers were appalled by the tragic movement of those

who attempted to escape from the area of famine: "All cities are crowded with these refugees awaiting transportation; the floors of [railroad] stations are covered with persons and their goods. The overflow camps on station platforms or on streets beneath eaves of buildings, or under standing freight cars for days and nights awaiting trains" (*ibid.*, p. 11). There seemed to be no system, no clear goal in their movement; there appeared to be as many coming as going. Many children were lost in the confusion, and a great many others were deserted by their parents, who were unable to provide them with food.

The picture of the horror of starvation, as seen by the American Relief Administration workers in 1921-22, was summarized by Fisher (1927, esp. pp. 85-98). In the starving villages "not a living soul could be seen in the streets. . . . To find a quarter or even half of the houses closed and boarded up, attesting the flight of their occupants, was common. Already the deaths from starvation were so many, that village clerks no longer kept records. . . . Of those who remained alive, nearly all were subsisting on food substitutes, the components of which indicated the degree of their want. The more fortunate mixed grain with chaff or ground weeds and acorns. Others, having no grain, made nauseating, poisonous concoctions of weeds, tree bark, and even clay and manure. Such domestic animals as there remained were fast disappearing. They starved and died like their owners, or were killed for food. Men and women in desperation exhumed dead animals, or killed and hungrily devoured cats and dogs when they could be found. . . . To the peasant crazed with hunger, who had come to eating the flesh of animals dug up from the ground, the practice of eating human flesh was not such a long step" (*ibid.*, p. 98).

In some instances cannibalism was preceded by murder, the flesh being made available for public consumption through somewhat irregular business channels. Fisher records an incident in the city of Orenburg where a head was found in the streets, and the investigation "discovered that the murderer had cut up the body of his victim and sold the flesh to a Persian, who in turn sold it in the bazaar. This case resulted in the issuance of an order by the city authorities forbidding the sale of meatballs, cutlets, and all forms of hashed meats" (*ibid.*, p. 109). The report of the medical division of the American Relief Administration quotes the work of Professor Frank of the Department of Mental and Nervous Diseases of Kharkov University, who investigated a number of cases of cannibalism: "He was able to establish the authenticity of twenty-six cases in which humans were killed and eaten by their murderers. He found seven cases in which murder was committed and the bodies sold for pecuniary gain. In these latter the flesh was disguised in sausage form and placed on the open market. The practice of necrophagia he found very common in all districts" (*ibid.*, p. 436; see also Frank, 1923).

The psychopathology of extreme hunger in the years 1921-22 was discussed in detail, with a documentation of the cases of cannibalism, by Rozenstein (1926). With reference to the occurrence of this phenomenon Rozenstein noted that in the district of Samara there were hundreds of thousands of hunger deaths; the cases of cannibalism amounted to only a few hundred, and the instances of murder and cannibalism could be counted only in terms of dozens. In

the psychiatric hospital of Samara. Rozenstein examined 35 patients, including 16 children, who had eaten human flesh; 3 of the adults had murdered their victims. The murderer-cannibals were of very low grade intelligence and their personalities were described as "primitive." Even after 4 to 5 months in the psychiatric hospital all these patients showed symptoms of severe physical exhaustion. The resort to human flesh, often after months of ever-increasing hunger pangs, appeared to be an animal-like reaction without painful emotional overtones.

Observations in Germany—World War I

After 3 years of undernutrition during World War I the children in Trier, Germany, showed decreased physical and nervous energy, heightened irritability, and emotional changes which were considered characteristic of neurasthenia (Blanton, 1919). Although their measured intelligence was not affected, they did poor work in school and the lassitude manifest in their general behavior often made them appear stupid. Teachers said the children grew tired more quickly than during prewar days, that they were able to pay attention for only a short time, that their comprehension was slower and their memory poorer, that they were restless, and that discipline was harder to maintain. Playtime had to be reduced and running games and hikes discontinued because the children lacked physical endurance. They tended to become sleepy during school hours and to require more sleep at night.

German adults who were similarly undernourished presented a similar picture. They complained of poor memory, lack of ability to concentrate on mental work, reduced physical energy and work capacity, and nervous exhaustion (Fischer, 1923). Rubner (1928, p. 78) mentioned the deterioration of performance by industrial workers and hospital personnel. The professional people—doctors, judges, ministers, teachers—as well as clerical employees complained of lack of endurance in their work, difficulty with arithmetic, and forgetfulness. However, more striking than the deterioration of capacity for intellectual work was the decrease in individual initiative, the apathy toward important events, indecision, and a depressed mood. In another context Rubner (1928, Vol. II, p. 20) recorded the feelings of hunger, tiredness, and chilliness. Nocturia in adults and bed-wetting in children were ascribed to the watery diet and to the decreased sensitivity of the central nervous system to the stimuli which indicate the need for voiding the bladder. Some observers reported a decrease of the *libido sexualis*; on autopsy a pronounced atrophy of the testes was noted. There was an increase in the number of patients with neurasthenic symptoms, but the incidence of mental diseases was not altered. Brugsch (1928, Vol. I, p. 277) included among the characteristics of undernourishment and semi-starvation a heightened irritability, polyuria, dizziness, headache, and sleepiness.

Famine in the Warsaw Ghetto, 1941-1943

The large Jewish community in Warsaw had the tragic privilege of being the first in the course of World War II to be subjected by the Germans to severe and prolonged semi-starvation. The medical aspects of this "natural" semi-star-

vation experiment were discussed by a group of physicians, almost all of whom perished before or during the complete destruction of the ghetto, ending the desperate, month-long (April 19 to May 20, 1943) Battle of the Ghetto (Apfelbaum, ed., 1946).

The psychological observations, limited to hospitalized patients, were made only incidentally. Fliederaum *et al.* (1946, pp. 110–11) spoke of bradyphrenia, a pronounced slowing down of mental functions. The processes of association were sluggish, the mental content was impoverished, the interests narrowed down. Young people, once full of life and energy, became apathetic and depressed, lost initiative, and would lie down to sleep whenever possible. The passage from life to death was gradual, as in extreme old age, and often it was difficult to recognize when the death occurred. As one of the patients put it: "The strength melts away as the wax of a candle." Life was extinguished like the light of a lamp which runs out of oil.

The Siege of Leningrad, 1941–1942

The German blockade of Leningrad in World War II produced a food shortage that rapidly resulted in famine in the winter and spring of 1941–42. There were also other causes of distress—the strain of bombardment, destruction of shelter and community facilities, excessive demands for physical effort, and so on. The available reports deal only with conditions as seen in the hospitals (Tushinskii, Aleshina, and Zeits, 1943; see also Brožek, Wells, and Keys, 1946, and Brožek, Chapman, and Keys, 1948).

Drastic cuts in food rations began in November, and in about 4 weeks emaciation and decreased physical strength became noticeable; numerous cases of people fainting at work came to the attention of the First Aid Service. In general, the signs, symptoms, and complaints conformed to the usual picture of famine. In a sample of 42 patients prominent complaints were polyuria (85 per cent), aches and pains in the extremities (85 per cent), sensitivity to cold (80 per cent), diarrhea (75 per cent), and dryness of the skin and hair (80 per cent); only one patient did not complain of fatigue, and in only one case was there a loss of appetite.

Psychologically, various degrees of deterioration were observed. This was expressed as apathy, dulling of the emotions, lowering of the moral level, narrowing of the intellectual interests, and obliteration of the individual characteristics of the patients (Chernorutskii, 1943, p. 5). Psychotic manifestations, hallucinations, and delirium were rare even in patients who otherwise exhibited the classical signs of pellagra with dermatitis, glossitis, and diarrhea.

The peak of the incidence of pellagra was reached in May and June of 1942, that is, after some 6 months of severe food shortage. Out of 42 pellagrins only 2 showed severe psychopathological symptoms. The other 40 patients did not appear to differ psychologically from the patients with simple semi-starvation, sharing with them a certain inhibition of psychological reactions, depressed mood, and neurasthenia (Khvilitskaia, 1943, p. 69). The absence of the dementia so common in ordinary pellagra was considered to be a result of the more rapid development of the illness as compared with endemic pellagra.

Famine in the Netherlands, 1944-1945

After 4 years of moderate food shortages, real famine developed in western Holland near the end of World War II (October 1944 to May 1945). Conditions were worst in the larger cities. In an affected population of under 6 million, there were, by direct count, many thousands of excess deaths (see Dols and van Arcken, 1946; Boerema, 1947). Nutritional survey teams which went into the area immediately after liberation found about 10 per cent of the active (street) population showing signs of serious undernutrition (Burger, Sandstead, and Drummond, 1945). Common complaints were physical and mental exhaustion, dizziness and a tendency to collapse after long standing, backache and aching legs, numbness and tingling of fingers and toes, sensitivity to cold, polyuria, and diarrhea. Psychoses secondary to starvation were also reported. In some cases this was thought to be due to hypoglycemia and the condition responded well to intravenous administration of glucose (*ibid.*, p. 283); rapid improvement was observed also when forced feeding was used.

Throughout the German occupation, including the period of famine, the Dutch organization of government and community services was admirably maintained, and there was no mass hysteria or major social disintegration. The underground was constantly active and effective against the Germans, but ordinary civil crime presented no real problems. At the time of liberation the transition from German rule to their own governmental machinery was smooth and relatively uneventful. The firm organization of both civil and military police acted as a deterrent, to be sure, but it was clear that the Dutch never lost their sense of discipline and political unity.

In spite of the "good" behavior of the Dutch in their severe tribulation, there were signs of abnormal behavior. Meerloo (1945) mentioned irritability, nervousness, and hostility. Among hospital patients there was a general appearance of mental dullness; questions had to be repeated and answers were given slowly. For the most part the patients were silent and apathetic. There was emotional depression, but the Dutch physicians emphasized that one could not speak of a "hunger psychosis" (Boerema, 1947, p. 235). The Dutch observers noted general sexual depression. Libido was diminished or absent; flirtation was conspicuous by its absence; no sex jokes were heard. Among women menstrual disturbances, particularly amenorrhea, were widespread.

In respect to the more severely starved individuals the Dutch observations are in full accord with all other reports of famine: "The most striking symptom of starving persons is their total apathy, in spite of the fact that they often had lost their self control and were most irritable" (Dols and van Arcken, 1946, p. 354). In the terminal stage of starvation the Dutch patients appeared to wish only to be left alone and did not ask for help or search for food.

At the peak of starvation "People dropped from exhaustion in the streets and many died there. Often people were so fatigued that they were unable to return home, before curfew; so they hid in barns or elsewhere to sleep and there died. Older people, who lacked the strength to go searching for food, stayed at home in bed and died" (Burger, Drummond, and Sandstead, eds., 1948, p. 22).

Members of Allied Medical Feeding Teams noted the tendency of the semi-

starved Dutchmen to dodge issues rather than to face them squarely. The return toward clarity of thought and briskness of decision and action, following within a month of refeeding, provided a striking contrast to the listlessness and indecision of semi-starvation.

Effect of Semi-Starvation on Patients in a French Mental Institution

Although this monograph is concerned primarily with the changes, including those in personality, which take place in previously normal individuals during semi-starvation, the effects on symptomatology and the course of diseases are also of interest. In some instances the effects of reduced food intake are actually beneficial; for example, during the famine attendant on the siege of Leningrad in 1941–42 there was a significant improvement in the clinical picture of existing hypertension (Brožek, Chapman, and Keys, 1948). Amelioration of diabetes is another and perhaps more striking instance of a positive effect of semi-starvation (see Chapter 48). On the other hand, the aggravation of the course of tuberculosis and the increase in its incidence illustrates the deleterious effects of caloric reduction on health.

Information on the effect of starvation on existing mental diseases is scant, even though during World War II the food intake of the inmates of the mental hospitals in Europe was often severely restricted. Bachet (1943, esp. pp. 204–6) reported that in one French institution semi-starvation produced no observable changes in the mental status of the patients that could be related to the deterioration of nutritional status. This was true also of those mental patients who suffered from nutritional edema, diarrhea, and pellagrous erythema. In manic depressives neither the rhythm nor the severity of the two phases of the psychosis was modified. The recovery of patients with anxiety neurosis, depression, and melancholia proceeded at the normal rate during the period of nutritional restriction. Bachet pointed out that there were changes in the behavior of the patients, but these changes were ascribed solely to physical weakness and not to a superimposed psychopathic state.

In evaluating Bachet's work it should be kept in mind that his conclusions were based on gross clinical observations and impressions and that no indications were given concerning the number of patients observed.

Semi-Starvation in European Concentration Camps and Camps for Prisoners of War

In Chapter 36, which introduced the chapters on the psychology of starvation, the limitations of observations made in the various European prison camps during World War II were stressed. Nevertheless, any account of semi-starvation in the 20th century would be grossly incomplete if it did not consider the experiences of the hundreds of thousands of men, women, and children who suffered and many of whom died because of the inadequacy of food. The material is abundant, and no attempt is made to cover the literature exhaustively. As illustration, the plight of the Spaniards interned in southern France and the psychological conditions of the inmates of the Belsen concentration camp in Germany are described in some detail. Observations on conditions typical of the

camps for prisoners of war in Germany are also presented. The numerous reports from the Far East are of only limited interest because the picture of caloric deficiency was severely complicated by vitamin deficiencies. In all cases only technical reports made by medical officers are included. The growing volume of memoir literature, even though it is not without psychological interest, cannot be considered here.

Interned Spaniards in France, 1941-1942

Conditions in the internment camps in southern France, the population of which consisted predominantly of the Spaniards who fled their country at the end of the civil war, were described by Zimmer, Weill, and Dubois (1944). The severely cachectic patients exhibited a marked muscular hypotony, and their posture was poor. They had difficulty in standing up and in keeping their heads steady. After standing a short time — a matter of a few minutes — they experienced pricking pain in the limbs, began to tremble, sometimes sweated profusely, and fainted. The patients in a progressed stage of semi-starvation were extremely weak, tended to drop things they held in their hands, and finally were not able to stand up at all, their only means of getting around being to creep on all fours. In general, men appeared to be less resistant to semi-starvation than women; more of them showed bradycardia, lowered blood pressure, and ascites. All suffered from extreme fatigability. The body temperature was low, and the patients were very sensitive to cold. General sensitivity was said to be normal. There was no deterioration of hearing. Some cases of night blindness were observed, and several cases of precocious presbyopia were reported. In some patients paresthesias and polyneuritic pains, as well as ataxia, were present, and the knee jerks were absent.

The therapeutic effects of thiamine and brewer's yeast were regarded as evidence that the neurological signs resulted from a deficiency of vitamin B complex. In severe cases of semi-starvation the gaze was dull and the patients were apathetic; their answers to questions were monosyllabic and were given only after a noticeable delay. The slowness of their mental processes continued to be apparent even during convalescence. Patients who were in the last stages of starvation, weakened to an extreme degree and collapsed on the straw, exhibited mental confusion and amnesia; a spasmodic laughing or crying was sometimes observed.

A few details may be filled in from Schwarz's (1945) report. He commented that the facial expression was painful (*facies dolorosa*), sometimes masklike. There was an acute desire for food in large amounts, and frequently the patients had to be torn away from the refuse cans. The heightened desire to eat tended to last long after the patients became well. Even in the treatment of edematous patients the food had to be mixed with large amounts of liquid to make up the quantity they desired. The daily bread rations had to be divided into three portions and given out separately; otherwise all the bread would be eaten as soon as it was received. Yet in spite of the intense craving for food even some very sick patients would trade bread for tobacco, and their bread had to be incorporated in the meal.

Loss of appetite was an ominous sign; it appeared to be related to cessation of the secretion of digestive juices and frequently indicated the approach of the terminal stage of semi-starvation. Even though the use of hydrochloric acid and pepsin frequently seemed effective and the patients began to eat again, the final outcome was repeatedly fatal. There was a tendency to constipation, although diarrhea was occasionally present. The universal polyuria was explainable by the large amount of liquid in the camp diet and ceased promptly when the food improved. On the other hand, nocturia persisted for a considerable length of time. Except for exhaustion and changes in reflex responses, neuropathological changes were observed rarely and only in the later stages of semi-starvation. At a late stage the patients developed a psychosis marked by hallucinations, and then the changes were, as a rule, irreversible.

When additional food supplies were made available, there was a gradual return toward normal in appearance and in state of health, and the atmosphere of doom and desolation gave way to a more optimistic outlook as the patients gained in weight and strength.

Belgian Civilian Prisoners, 1940-1944

The effects of semi-starvation in civilian prisoners, completely dependent on the official food rations, were studied by Simonart (1948). Before the war the average daily food intake in the central prison of Louvain, Belgium, amounted to 3370 Cal. In October 1940 it was reduced to 1775 Cal.; the values for March, May, and July 1941 were 1860 Cal., 1666 Cal., and 1647 Cal. Edema, which is the main topic of Simonart's study, appeared within 3 months on the semi-starvation diet and was present in July 1941 in 39 per cent of the prison population; in one extreme case 20 liters of edema fluid were found (patient V, p. 49: pre-starvation weight, 70 kg.; weight of body plus edema, 74 kg.; after diuresis, 54 kg.).

The inmates suffered from severe hunger pangs much of the time. They consumed their day's entire allotment of bread (225 gm.) at breakfast (*ibid.*, p. 69). To allay their sharp and ever-present hunger they ate vegetable parings, potato peelings, carrot tops, and even decayed leaves of cabbage. A prisoner working in the garden was seen to suck on a bone retrieved from a garbage can. Another man mixed sawdust into his soup to thicken it. Still another prisoner ate a bird, raw, which he caught during exercise in the prison courtyard. Because the sensation of hunger is related by some authors to the secretion of hydrochloric acid in the stomach, it is of interest to note that in 8 out of 10 semi-starvation patients who were examined the secretion was impaired or absent, even after injection of histamine; yet they suffered sharp and persistent hunger pangs.

The frequency of urination was increased and nocturia appeared (average urine output in 3 patients was 2.75, 3.37, and 2.12 liters during the day and 4.05, 3.86, and 3.69 liters during the night). The patients felt weak, showed numbness and paresthesias. Constipation was mentioned as one of the semi-starvation symptoms (*ibid.*, p. 72), bowel movements being reduced from once a day to one or two times a week. The men were living under relatively good hygienic

conditions. In the majority of field reports diarrhea rather than constipation received prominent mention. It was noted only occasionally in Simonart's material and was entirely absent in the semi-starvation phase of the Minnesota Experiment.

Simonart (1948, p. 145) noted that during nutritional rehabilitation the semi-starvation asthenia was only slowly replaced by normal vigor and the urinary disturbances (pollakiuria, polyuria, and nocturia) were among the most persistent symptoms. Paresthesias and numbness and changes in skin sensitivity and tendon reflexes of the limbs disappeared early in the course of the treatment. In Simonart's experience the time necessary for the restoration of normal physical status varied between 3 and 12 months.

The Belsen Camp, 1945

The psychiatric manifestations of severe semi-starvation in the Belsen concentration camp, taken over by the British Army in the second half of April 1945, were described by Lipscomb (1945). The diet was always inadequate, but in the winter of 1944-45 it became grossly deficient. It may be useful to quote a part of the report verbatim: "The most conspicuous psychological abnormality was a degradation of moral standards characterized by increasing selfishness, and it was more or less proportional to the degree of undernutrition. In the first stage consideration for others was limited to personal friends, then the circle contracted to child or parent, and finally only the instinct to survive remained. Emotional response became progressively lowered and consciousness of sex was lost. Eventually all self-respect disappeared and the only interest left was to obtain something which could be eaten, even human flesh. Even among those not grossly under-nourished there was a blunting of sensitivity to scenes of cruelty and death" (p. 315).

More detailed psychological investigations by means of interviews of a group of internees who had lived through the terrible starvation experience at the Belsen concentration camp were carried out by Niremberski (1946). His main task was to examine patients who had been referred to him by other physicians for psychiatric attention. In addition, in the hospital and in the convalescent areas he examined some 60 patients who were selected at random and who were taken to represent a cross section of the camp population. Unfortunately, in the description the information from these two sources is not clearly differentiated.

At the time of liberation the camp was estimated to contain about 50,000 people, with some 10,000 lying dead in the huts and around the camp (see also Mollison, 1946). There was marked reduction in activity, sometimes reaching complete immobility. Many internees did not have strength enough to walk. A common reaction was the loss of appreciation of social ties, each individual living entirely by himself; family had little meaning. The responsiveness to death, cruelty, and humiliation was blunted, and the individuals became apathetic to incidents in the camp. Many showed discouragement, seclusion, introversion, and depression. Only a minority exhibited aggressive reactions. Impairment of memory appeared common. Carelessness about appearance, particularly in men, tended to persist even after liberation. In women the sense of modesty was dull

or lacking. The interests were severely narrowed down to the question of food. The inmates were continually hungry, and in the last period of the camp before liberation cannibalism occurred.

The cases with psychopathologies were few in view of the harrowing experiences, the physical suffering, and the fear of hunger, torture, and death to which the internees were exposed. There were cases of conversion hysteria, expressed in the inability to hear or see. Delusions were rare. A few patients exhibited maniacal outbursts.

Observations on Former Inmates of German Concentration Camps — Switzerland, 1945

In the spring of 1945 two groups of former inmates of German concentration camps, mostly deportees from occupied Allied countries, found refuge in Switzerland, one in the military hospital at Herisau, the other in the Cantonal Hospital at St. Gallen. The groups numbered 296 and 57 patients, respectively. The psychological observations are scattered throughout the monograph that reports the studies made on these patients (Hottinger *et al.*, 1948).

The patients who had been exposed to prolonged and profound semi-starvation exhibited the classical starvation syndrome (severe muscular atrophy, weakness, edema, polyuria, bradycardia, hypotension, and hypothermia), together with diarrhea, secondary infections (especially those of the skin), and tuberculosis. The psychological status on arrival was described as catatonic and infantile. There was a marked apathy. A general slowing down of behavioral reactions, complaints of poor memory, some night blindness, failure of the extended pupils to react to light, changes in taste (*ibid.*, p. 31), paresthesias and dysesthesias in the extremities, uncertain movements, tremor, diminution of skin and tendon reflexes (p. 32), extinction of libido, egocentricity and drowsiness (p. 126), narrowing of interest to questions of food (p. 149), and fatigability (p. 210) were noted. There were 3 cases of hysteria, 1 of schizophrenia, and 3 of misbehavior (*Trotzneurosen*); these were described in some detail (pp. 91-95).

Ex-Prisoners in the Hospital at Mainau, 1945

A group of 40 severely starved French ex-prisoners of war were studied in detail by Lamy, Lamotte, and Lamotte-Barrillon (1948). On arrival at the hospital in Mainau they were profoundly cachectic, the average weight loss being 38.4 per cent for 19 patients for whom the pre-starvation weights were available. They would lie immobile, indifferent to their environment, asking no questions. During interview they spoke slowly, in a monotonous tone, and related their experience in a detached manner. There was no impairment of intelligence. Their judgment appeared intact, and their answers were rational and precise. On the other hand, they had difficulty in maintaining attention for any length of time and the interviews had to be carried out in a number of short sessions. Memory was in general well preserved. These individuals, many of whom had been deported to Germany for participating in the French resistance movement, lost in the detention camps their interest in political questions and became concerned only with the immediate problems of the internment life and the possi-

bility of escape. In the last stages the men were overcome with apathy and resignation. Even life itself mattered little.

In the course of 3½ months of hospital care the interests of these men progressively widened. Those who had been totally indifferent on arrival began to show an interest in food, their health, the time needed for recovery, and the fate of their families. In two weeks many were asking for papers to read. Yet initiative was slow in returning, and fatigability and inability to concentrate tended to persist.

Leyton's Observations on Prisoners of War — World War II

G. B. Leyton, as a captured medical officer, had unusual opportunities to observe the effects of slow starvation in German prisoner-of-war camps in Libya, Italy, and Germany. He made systematic observations and preserved his records, which eventually made a notable M.D. thesis at Cambridge University.

Leyton (1946) summarized the reactions of the soldier to a sudden reduction in diet: "First there was a loss of the natural feeling of well-being. A growing feeling of hunger followed and gradually increased in intensity until, after about 3 weeks, the whole thought of the prisoner-of-war was concentrated on his food. His chief concern was how long it was to the next meal, and by what means he could supplement the meagre rations. This insistent feeling of hunger remained after years of a low diet. It appeared even to increase with time, with the result that the half-starved man would go to the greatest lengths of ingenuity and dishonesty to obtain small amounts of extra nourishment. Only when death was imminent did the desire for food slowly vanish and the grossly emaciated prisoner become resigned to his fate, of which he seemed to be aware some weeks before it happened.

"The next notable abnormality was a progressive mental and physical lethargy. The desire for sleep would increase; the number of hours that an adult male would wish to remain in bed, partly dozing but for the most part in genuine deep sleep, steadily rose from the normal 8 hours to 16 or more out of the 24. Finally, the only way to rouse a man from his bed was by the mention of food. Shortly after this came the rapid fatigue in mental and physical effort. The experienced card-player, for instance, would forget the cards that had been played. Part of this was certainly lack of power of concentration, but the major portion was a lack of memory for recent events, though the memory for distant ones was still normal" (*ibid.*, p. 75).

The first definite signs of pathology appeared after insatiable hunger and marked lethargy had been present for some days or weeks. In the fully developed picture of semi-starvation the slowness of movement was notable. The gait in walking was a shuffle and suggested a slow-motion picture. In the X-ray room the men executed the movements only slowly and after a long delay when told to place their hands on their heads. Speech was slow, and obtaining responses to questions was tedious. Leyton thought there was exceedingly slow cerebration, but the evidence he mentioned could be taken equally well to indicate only apathy and reluctance to talk.

The prisoners were kept for long hours at physical work in spite of advanced

undernutrition. Their slow movements and delayed reaction times made these men especially prone to accidents: "Though they could see that a fall of rock or branch of a tree was imminent, their actions were too slow to move out of danger" (*ibid.*, p. 79).

All thoughts became centered around food. Sexual desire was reduced but only rarely did it disappear completely; in a few patients who appeared to be in the last stages of semi-starvation it seemed intensified. Interest in such recreational pursuits as reading and playing games all but disappeared. The desire for alcohol was not affected. The desire for tobacco greatly increased and appeared to be stronger than the desire for food, since the men would barter even the little food they had for a small amount of tobacco. Standards of cleanliness deteriorated drastically, and pride in personal appearance was lost. Normal moral standards lost all influence on the behavior of the starving men, and they would steal food from their best friends or barter their clothes for tobacco. "None of the other hardships suffered by fighting men observed by me brought about such a rapid or complete degeneration of character as chronic starvation" (*ibid.*).

Other European Prison Camps — World War II

Many accounts of starving prisoners in German camps in World War II have some limited interest in providing variations in the description of essentially constant phenomena. In general, the picture was one of inadequate clothing, grossly deficient diet, forced labor to an excessive degree, brutality, and callousness. Whether by accident or design, the result of the semi-starvation imposed on the prisoners was that they were easily managed; their apathy made them docile.

Stevenson's report is in agreement with the findings of Leyton (see above). The British prisoners of war were depressed and lost all sex interest. They "felt confused in their minds and were unable to concentrate on things like simple games and books" (Stevenson, 1944, p. 659).

Markowski (1946) gave a vivid description of a scene at the German camp at New Brandenburg: "[The dinner] was composed of a watery vegetable soup doled out once a day in meagre quantities. It was winter. I saw a long line of terribly emaciated Russian prisoners in rags. Their feet, blue with cold, were sometimes bare, and sometimes they wore wooden clogs. They held their mess-tins in trembling hands, numb with cold. But it was their eyes that drew my attention. They had nothing human in them; they were deeply set, shining, and fixed with an animal expression on the soup-container. They seemed to see nothing except it; they seemed not to notice that some of their comrades had fallen from exhaustion and that they were treading upon them, doing them to death by the trampling of their clogs. Their only idea was to get nearer to the soup-container and obtain their ration quickly." There was definite evidence of cannibalism in this camp, with pieces of flesh cut out of the dead bodies.

Japanese Prison Camps — World War II

Conditions in the Japanese camps resembled those in the German camps, but there were some peculiarities which probably reflected the fact that in the Far

East the diet was apt to be more deficient in specific nutrients. For example, of 2734 repatriated ex-internees from the Santo Tomas Camp in the Philippines, about one fourth had signs that would be attributed to vitamin deficiency disease (see Butler *et al.*, 1945; Musselman, 1945).

Reports on behavior and psychological manifestations in the Japanese camps are few and are confused with descriptions of purely physical symptoms. Fatigue, apathy, depression, irritability, and loss of libido were noted in the Far East as elsewhere but were not documented or systematically examined. Musselman (1945) made the interesting statement that an increase in food, resulting from the arrival of Red Cross packages early in 1943, produced an increase in libido as expressed in the frequency of erections and ejaculations.

We have been able to interview a number of repatriated ex-prisoners, both military and civilian, of the Japanese. Their accounts have the weakness of being recollections after, in many cases rather long after, the fact. But a number of points seem to be verified from independent witnesses and may be offered here. Perhaps the first item of emphasis is the wide diversity of behavior in all except the most extreme stages of starvation.

In the Japanese camps there was little if any deliberate attempt to produce starvation death; the diet was often extremely deficient, but this was the result of Oriental dietary standards plus dishonesty in the local administration. The Occidentals, with their larger bodies and higher metabolism, suffered far more than the Japanese would have on the same diets. In any case, the debility from pure caloric deficit was generally less than in the corresponding German camps. Under these circumstances of severe but not completely intolerable food shortage life went on for years in these camps. Some individuals rapidly deteriorated psychologically, but others made surprisingly good adjustments and "found" themselves in various group enterprises. There are individuals who actually look back with nostalgia on some aspects of their prison experience; these are persons who discovered and used their capacity for leadership in the camps.

In almost all cases there was a narrowing of social interests; the world outside the camp receded into relative unreality. There were innumerable instances of hoarding and petty dishonesty, but in only a few cases was there any readiness to disregard totally the welfare of the group for personal advantage. All personal belongings, but especially food, were defended fiercely, and distrust was easily engendered. Quite generally the custom developed of eating in privacy, partly to avoid any need to share little extras, partly to indulge in the taste sensation without distraction. In several cases this custom was so firmly fixed that the liberating American troops had real difficulty in establishing temporary communal mess halls.

Observations on Chinese Repatriates—World War II

Laycock (1944) reported observations on refugees and repatriates who were transferred from occupied into free China in 1942-43. The Japanese first placed the repatriates in concentration camps, with rice gruel (without salt or flavoring) as the only diet. Later, in a state of exhaustion, they were transported to the coast of free China. Laycock paints a sorry picture: "The ones who are too weak to walk remain where they are landed until they starve. Many of them try

to prolong their suffering by consuming inedible or semi-edible substances. I have been shown hard woody roots and have been told that after prolonged soaking these can be cooked into something that can be swallowed. . . . The refugees also steal and eat unripe rice, a crime for which the penalty is death. . . . I am also convinced from the evidence of eye-witnesses that the flesh cut from corpses is used as food. Why more of these unfortunates do not give up the unequal struggle and find an easy way out in suicide I do not know. Certainly very few of them seem to make the attempt. Instead they struggle inland with a frantic desperation of which one is forcibly reminded by seeing the deep ulcers on the buttocks of people too weak to walk who have struggled across the country in a sitting posture" (p. 667). Many complained of numbness in the feet and legs but anesthesia to pin prick, paresthesias, and paralyses were uncommon. The dominant emotional state was one of depression. The children would lie curled up all day, motionless, and their edematous skin acquired deep impressions from the straw mats. In the adult patients cerebration seemed to be slow, and the extraction of the medical history was a tedious procedure.

Episodes of Starvation among Marooned American Military Personnel

During World War II there were many instances when men had to abandon ship or parachute from an airplane and then were forced to subsist for days or weeks on grossly inadequate diets. Undoubtedly in many cases no survivors lived to tell the tale, but the number of rescues after shorter or longer periods of survival is impressive. The saga of the survival of U.S. Navy personnel who were forced to abandon their ships and planes was briefly told by Lieutenant Commander Harby (1945). The accounts so far released stress courage and ingenuity, the tales of heroism that are associated with military purposes during or soon after war. Valuable materials, still largely inaccessible in the military archives of the various powers, await future objective analysis from which more valid generalizations can be drawn than from a few selected human interest stories. But the latter, like the accounts of explorers, have their validity as demonstrations of what man *can* do.

Lieutenant Leon Crane

On a routine test flight from Ladd Field, Alaska, Lieutenant Leon Crane's (1944) plane suddenly went out of control at an altitude of 20,000 feet, and the crew was forced to bail out. With his parachute he landed on a mountainside; with no gloves, with night temperatures of -40° to -50° F., and with no food or weapons his situation was indeed precarious. His whole "outfit" consisted of a parachute, 2 pads of matches, and a Boy Scout knife. Following a stream, he started his trek back to civilization.

After a cold and weary night he resumed his journey. Walking through the deep snow was heavy labor, and he was growing very hungry. He wrote: "Toward afternoon I felt such a gnawing at my stomach that I scooped up snow in my hand, held it in a ball until it began to melt, and ate it, letting it melt against the roof of my mouth. I thought it helped a little." Toward evening he was able to satisfy his thirst by drinking from an overflow in the ice where the water was

not frozen. He found a good place for making camp and remained there for 4 days, hoping for a rescue party to spot the SOS marks he had made on the snow with pine boughs. On Christmas Day, the fourth morning of his adventure, he woke up with terrible hunger pangs. He thought of the milkshakes he would have at Ladd Field, and the snow melting in his mouth actually began to taste like a milkshake. He was unable to get hold of any food whatever. There were squirrels around but they remained out of reach. On the fifth day the pronounced sensations of hunger disappeared. His thirst, however, remained, and he drank a large amount of water. On the ninth day he resumed his journey and was fortunate enough to find an abandoned trapper's cabin which contained some food. However, a handful of raisins and 2 cups of hot cocoa was all he was able to eat on that day.

Although suffering from frostbite, Crane started on his journey again the next morning with a handful of raisins in his pocket. He walked all day and far into the night, until he became so weak he decided to return to the cabin, which he finally reached, utterly exhausted. By the afternoon of the third day he felt rested, though still weak, and was feeling hungry again. Searching the place more thoroughly, he found a considerable amount of food, as well as clothing, bedding, and a rifle with boxes of ammunition. At first he gorged himself with food but later he limited himself to two meals a day. He slept most of the first week and felt cold even though there was a fire and he had on plenty of clothes. Later he worked out a satisfactory routine, including hunting, and remained in the cabin until the middle part of February, when he started off, with food packed on top of a homemade sled, for the Yukon River. He reached an inhabited cabin early in March and was picked up at Woodchopper by a plane which brought him back to Ladd Field.

Eighty-Three Days on a Life Raft

The experience of Seaman Izzi, who drifted for 83 days on a raft in the Atlantic after his ship had been torpedoed and sunk several hundred miles off the coast of Brazil, was described by Murphy (1943). The Dutch luxury liner on which Izzi served as a member of the U.S. Navy armed guard went down on November 2, 1942. Only a handful of the more than 400 persons who were aboard the ship survived. The attack was sudden and damaged many of the lifeboats. Izzi and two other men of the gun crew were lucky to get hold of a little bamboo raft. Later they drifted near a larger bamboo raft and swam toward it. Other men also reached the flimsy raft, only to drift away after a while. "Maybe a guy thought of a better place to go, or maybe he got tired and quit, or maybe he just went to sleep. Then you would hear guys hollering for help, screaming that sharks are attacking them, and there was nothing you could do." Toward evening there were 2 men on top of the raft and 6 who clung to the side of it. About midnight Izzi began to have hallucinations, seeing repeatedly a stone wall and hearing the water lapping against it, and trying to swim toward it but being pulled back every time. Later he thought he was riding a motorboat and going to a night club across a lake; again he saw the wall as part of a lunchroom and was ready to set out for it.

Other men suffered from hallucinations too; two of the men left the raft to go over a hill nearby to get food. A day passed and another frightful night. On the following day Izzi got onto a substantial wooden raft, 8 feet wide and 9 feet long, already occupied by 4 other men, a U.S. Navy ensign, two Dutch seamen, and an American sailor. The raft was provided with such essentials as a medical kit, some rope, two paddles, a big canvas tarpaulin, etc. The food consisted of 9 cans of milk, a few dozen hardtack biscuits, a 2-lb. can of chocolate squares, and some 10 gallons of water. The men decided that a daily individual ration would consist of a cup of water and a piece of cracker for breakfast; a half cup of water, a little milk, and a piece of cracker for lunch; and a cup of water, a piece of hardtack, and a little piece of chocolate for supper. They stood watch day and night, each taking his turn.

The hot sun was a source of great discomfort, causing sunburn which developed into sores. During the first few weeks the men suffered greatly from hunger. Izzi described their attitude toward food: "We all waited for night to come so we could get our little piece of chocolate. When mealtime came around, we would act as if we were having a big meal, and we would try to make it last a long time, but it was usually over in two minutes." Both food and water ran out on November 18, the sixteenth day after sinking, but on the nineteenth day it rained and the water supply was replenished. Ensign Maddox caught a small shark with a lasso while Izzi's feet, dangling over the side of the raft, served as unintended bait. Maddox ate the heart and the other men consumed the liver; the rest of the meat spoiled over night. On Thanksgiving Day Hoogendam, a 17-year-old Dutch merchant seaman, caught a bird, the size of a chicken. In their 12 weeks of drifting they got some 25 birds, mostly small ones. The entrails were used for fishing. At other times the men tried to catch fish by hand through a hole cut in the deck of the raft.

By the end of the second month the rainy season began and the raging storms made life even more miserable. In order to improve their fishing facilities they broke apart the scissors contained in the medical kit, tied each blade to a stick, and used these for spearing fish. Much of the time they talked about food they liked or described what they were going to eat when they got ashore. Izzi said later: "That talking about food was good. You talk about it and you think about it, and your mouth gets watery, and jeez, it helps!" There was only a little talk about drinks and not much about women. It rained most of December. Three men had bad colds, and Beasley, the American sailor, became seriously ill. All were severely emaciated and very weak. When about the middle of December they spotted a ship, 2 of them had to hold the man who was waving the flag, and he could wave only 2 or 3 times before becoming exhausted. When the ship disappeared in the distance without taking notice of them, they were so discouraged they broke into tears.

The condition of the American sailor was getting critical. He became blind in one eye, then began to lose his hearing and the sight in the other eye, became delirious, and died the morning of the sixty-sixth day. Ensign Maddox was the next man to become ill, with similar symptoms, except for an added marked nervousness. When the water supply ran out on the seventy-sixth day, Maddox

had long spells of delirium and died that night. A stream of fresh water, into which they unexpectedly ran, saved them from probable death from lack of water, while snails found under the raft provided a badly needed new source of food. The three men who survived the ordeal were picked up by a Navy PC boat on the eighty-third day of their involuntary journey.

Starvation among Marooned American Settlers—The Donner Party

There are many incidents of starvation in which the lack of food was not due to a crop failure or the ravages of war. The history of the Donner Party may serve to illustrate one type of such emergency situations.

In 1846 a group of families traveling from Illinois to settle in California was marooned in the Sierra Nevada Mountains. Of the 81 persons who were overtaken by the snows, 45 died of starvation and exhaustion. They were fairly ordinary folk, inexperienced in the ways of explorers but courageous and resourceful. Contemporary records, diaries, and published recollections of the survivors provide a fairly full account of the behavior of a starving Occidental community lacking effective leadership and experience in coping with the catastrophe.

At first the interests of individuals and families prevented any realistic efforts to attack their problem as a group. They settled down in ignorance and attempted to allay their fears with unjustified hopes. The presence of many children and feeble adults made the whole group immobile. Later, break-through parties were organized in desperation, but sentiment and individual considerations determined decisions as to personnel. Discipline, which had never been developed in the group, vanished completely. What was a bad but not impossible situation became a major tragedy.

The inadequate food supplies consisted largely of meat from the cattle. Some animals were lost through carelessness in the first onslaught of the blizzard. The inexorable drive of hunger first made unusual foods acceptable and then led to attempts to eat the inedible. After weeks on very low rations and with 4 men dead in the main camp, "The little field mice that had crept into the camp were caught and used to ease the pangs of hunger. Also pieces of beef hide were cut into strips, singed, scraped, boiled to the consistency of glue and swallowed with an effort; for no degree of hunger could make the saltless sticky substance palatable. Marrowless bones, which had already been boiled and scraped, were now burned and eaten, even the bark and twigs of pine were chewed in the vain effort to soothe the gnawing which made one cry for bread and meat" (Houghton, 1911, p. 70). The same behavior is generally recorded in Chinese famines, where bark, weeds, and even dirt are eaten.

But the compulsion of hunger soon led to obvious expedients of greater moral extremity. The first of the break-through groups reached a settlement only after eating the flesh of their comrades who died on the way. Another group was caught by a storm in a break-through attempt and had to improvise what came to be known as the Starved Camp. Mr. and Mrs. Breen, left in this camp with 5 children of their own and 4 others, turned to the unburied dead to save those who were still living.

The diary of Mr. Breen, published by Teggart (1910), further documents

the use of human flesh in the entry for February 26, 1847: "Hungry times in camp. . . Mrs. Murphy said here yesterday that thought she would commence on Milt. and eat him. I don't [know] that she done so yet, it is distressing. The Downers told the California folks [of the relief party] that they commence to eat the dead people 4 days ago, if they did not succeed that day or next in finding their cattle then under 10 or 12 feet of snow and did not know the spot or near it. I suppose they have done so ere this time."

Under these circumstances the devotion of some individuals to their families was notable. Mrs. Donner chose to remain at the main camp by her sick husband to the end. When the final relief party got through to the camp, only one adult remained alive. He had sustained life by cannibalism and was suspected of having murdered the two remaining women.

The tragedy of the Donner Party is notable for the extent of recourse to cannibalism. It would be hazardous to attempt an explanation for this, but it should be remarked that extreme hunger persisted for months, that group discipline was practically nonexistent, that the bodies of the dead were preserved at hand by the cold, and that compunctions are easily overcome in the face of the needs of one's children.

Starvation and Hysteria—Shipwreck of Civilians

Shipwreck generally carries with it the shocking impact of sudden calamity, the change in a matter of hours or even minutes from relative security or even luxurious comfort to frightful hazard and a dreadful prospect. Without the numbing effect of a gradual decrease of food and physical strength, the passengers in a lifeboat may be overwhelmed by the very suddenness of the catastrophe and threatened doom. In the absence of a strong and immediately asserted controlling authority, all the elements of panic and hysteria are at hand. Once this tone is established, subsequent behavior in the absence of food is only partly determined by the effects of hunger itself.

Three instances in point were described by Weygandt (1904). In the case of the ship *Medusa* (1816) the physician Savigny reported that the survivors suffered from hunger pains but that exasperation about their situation was the most traumatic factor. Gross changes occurred in the personality of the shipwrecked, expressed in extreme irritability, suspicion, and cruelty. There were cases of murder and suicide, and both cadavers and excreta were eaten. Of 150 individuals only 15 remained alive at the end of their 13-day suffering. From the scanty data available it would seem that this group suffered a rapid and complete collapse of morale and discipline and that mass hysteria was more at fault than starvation itself.

The occurrence of visual illusions, observed in the shipwrecked passengers of the *Medusa*, was confirmed by the ship physician on the *Ville de Saint-Nazaire* (1896), who spent 7 days with 32 persons in an open boat on the high seas. Optical illusions are reported to have occurred by the second day and were followed somewhat later by auditory illusions, some individuals ending in delirium. The feeling of hunger was absent for the most part. Similar observations were made after the collision of the ship *Vaillant* (1897) with an iceberg; of 17

persons who managed to get in the boat, only 4 were found alive after 6 days. It was reported that by the second day the people were "losing their minds" and insisted on killing the dog, their only source of food, at a time when starvation had barely started.

Episodes like the foregoing are in great contrast to the ordinary experience of famine or even to misadventure and starvation among explorers. Though fear and foreboding may be prominent among all starving peoples, the most usual behavior is acceptance and resignation to a degree which seems remarkable to the well-fed observer. Apprehension about starvation is far more apt than starvation itself to provoke violent behavior. It is interesting to note that hallucinations may occur when death from starvation approaches, when there may well be an organic basis, or when the threat of starvation is presented and the trauma is largely psychic; over the major course of undernutrition and starvation such phenomena are absent.

Starvation among Polar Explorers

Exploration of remote regions always brings with it the chance of desperate mishap. In the heyday of polar expeditions in the 19th century, miscalculations and accidents exposed many exploring parties to months and years of short rations and outright starvation. It seems to be in the nature of explorers to keep diaries and to write accounts of their adventures; consequently there is a considerable literature containing factual reports of starvation among polar explorers.

The records of these unfortunate expeditions make fascinating reading, but their direct utility in the analysis of behavior in "natural" starvation is perhaps less than might be hoped at first sight. Often enough the violence of climate and terrain constituted tribulations as great as the food shortage, and men died from exposure and exhaustion as much as from starvation. Also it must be kept in mind that these explorers were no ordinary men. With few exceptions they were tough in mind and body and determined to remain so. They were schooled not to show their feelings, and in their reports they scorned to record the nuances of behavior. But the faithful day-by-day narratives of their existence convey significant factual details in spite of the characteristic laconic literary style.

The groups of men involved in the episodes noted in the following sections can certainly not be taken as representative samples of the general population. The ruggedness of their physical constitutions was remarkable, but their most extraordinary characteristic was their indomitable character. They exhibited an amazing capacity to resist the apathy and lethargy so characteristic of famine in more ordinary populations. These explorers showed how starving man *can* behave.

Franklin's First Expedition

Disaster met John Franklin's expedition (1823) on the return trip from the shores of the Arctic sea. The journey southward was started late in the summer of the year 1821, and as early as August 22 the meals were reduced to 2 a day. Numerous portages with heavy canoes weakened the men even when they occasionally were lucky enough to get fish and game. On September 7 Franklin fainted but after eating a little soup was able to continue through the half-frozen

swamps. A few partridges and a lichen called "rock tripe" had to suffice until a musk ox was killed on September 10. The contents of the stomach, followed by the raw intestines, were eaten on the spot before cooking the meat for the first real meal in a week. But this meat was soon gone and rock tripe again became the scanty and unsustaining diet. On September 22 a few pieces of skin and the bones of a deer devoured by wolves were discovered. The bones were burnt to make them "edible," and several men added their old shoes to the shreds of deer-skin for the meal.

Morale began to deteriorate and the men "ceased to dread punishment or hope for reward." Some of the scanty food reserves were stolen, and the hunters secretly ate some of the birds they shot. As debilitation progressed the sharpest pangs of hunger ceased, but food and the pleasures of eating were the central topic of all conversation. Two of the men became unable to go on and were abandoned to die. The next day the party was divided, Franklin and 8 others going ahead to seek help at Fort Enterprise. Four men of this forward party turned back and the other 5 reached the fort only to find it abandoned and devoid of food. Bits of deerskin, bones from the ash heap, and some lichens were all they could find. The 5 men stayed at the fort for some days before 2 of them, with only singed bits of hide as food, set out to look for succoring Indians. The little strength of the remaining men at Fort Enterprise declined so that only the greatest effort enabled them to rise from the floor, and a herd of reindeer half a mile away passed by because none could lift a gun.

Of the men who had been left in the encampment and of those who had turned back on the way to Fort Enterprise only the physician and one other man survived; they made their way to the fort, where they were distressed by the appearance of Franklin and his 2 companions. On the other hand, Franklin was shocked by the extreme debility of the 2 survivors and commented on the "sepulchral tone" of their voices. The survivors told how an Indian of their party had murdered 2 of the men. On one occasion this Indian had refused to go hunting and had remarked sullenly, "It is no use hunting, there are no animals, you had better kill and eat me." The Indian was stronger than the other 2 remaining men; he became arrogant and gave hints which caused alarm. When he appeared to be preparing to murder them, the physician shot him.

The 5 men at Fort Enterprise were now in a tragic state. Two of them died, and the survivors did not have enough strength to move their bodies. They had generalized edema but the acute pangs of hunger had subsided. Because of the loss of soft tissue, lying on the floor was extremely uncomfortable, but they dreaded having to make any movement. Under these conditions the frequency of urination, compelling the men to rise from bed 10 times or more during the night, was an extreme annoyance. It was attributed to the large quantities of "tea" they consumed. Their dreams often had a pleasant character, and often had to do with something about eating. Their minds began to wander, and each man most readily saw the mental weakness of his fellows. One of them (Hepburn) commented, "Dear me, if we are spared to return to England, I wonder if we shall recover our understanding."

On November 7 a relief party of Indians arrived with food. Although aware

of the danger of overeating, the men, including the physician who had repeatedly counseled moderation, ate a large quantity of food and suffered severe indigestion as a result. They were unable to control themselves. In contrast to the emaciated and feeble white men the Indians seemed gigantic and their strength supernatural. But good food worked wonders, and in 10 days the party set out from the fort with the Indians.

Greely's Adventures

One of the expeditions, the fate of which fired the imagination of contemporaries, was that led by Greely. An anonymous pamphlet published by Barclay and Co. at Philadelphia in 1884 was entitled *The Greely Arctic Expedition*, with the following subtitle: "Full account of the terrible sufferings on the ice, and awful tales of cannibalism." Greely's own account (Greely, 1886) is written in a matter-of-fact manner and does not mention cannibalism; on the contrary, in the dedication of the two volumes to the men of the Lady Franklin Bay Expedition he stressed "their loyalty and discipline in all the dark days."

The steamer *Proteus* failed to reach Lady Franklin Bay in the summer of 1883 to take Greely's men on board and the expedition was unexpectedly forced to spend the winter of 1883-84 in the far north. Rations were short by the end of October 1883. Lieutenant Lockwood's diary for this period, cited by Greely (*ibid.*, p. 176), states: "We are now in our hut, but it is not yet finished, and it is cold and uncomfortable. Our constant talk is about something to eat, and the different dishes we have enjoyed, or hope to enjoy on getting back to civilization. . . . We are all hungry all the time." In the entry for October 29 Lockwood says: "Occupied some time this morning in scraping, like a dog, in the place where the moulded dog-biscuits were emptied. Found a few crumbs of small pieces, and ate mould and all. We now get about one-fourth what we could eat at a meal, and this little ration is to be much further reduced" (p. 184).

The strain of semi-starvation was beginning to show in the increasing irritability of Greely's men. When the commander of the expedition decided to have all cooking done on one stove to save fuel, which necessitated dividing the men into two messes, the procedure was sharply criticized because half of the men had to wait until the other half was fed. The trust of the men in each other began to be undermined by such incidents as the disappearance of a package of tobacco. When Brainard, who was in charge of issuing food supplies, became ill, the cook was given the job of portioning out the rum. He took more than his share and was seen entering the commissary house without having legitimate cause to do so; on a later day a can of milk was found hidden away, the evidence pointing to the same man. It was an even more serious indication of social deterioration that the physician of the expedition should be accused of taking bread during the night from the bread can of a sick man. The entry of December 11 is indicative of the effects of semi starvation: "Brainard overworking himself again, and was faint and dizzy this evening. I was obliged to remonstrate for doing other work than issuing as I have forbidden it, but when he points out the apathy of the party, and the necessity, I am silenced" (p. 209).

Lockwood's diary brings out other aspects: "We are all very weak, and I feel

an apathy and cloudiness impossible to shake off. It is a great difficulty to know each night just how much hard bread to save for breakfast of the morrow — hunger to-night fights hunger tomorrow morning. I always eat my bread regretfully. If I eat it before tea, I regret that I did not keep it; and if I wait until tea comes, and then eat it, I drink my tea hastily and do not get the satisfaction I otherwise would. What a miserable life, when a few crumbs of bread weigh so on one's mind! It seems to be so with all the rest. All sorts of expedients are tried to cheat one's stomach, but with about the same result" (p. 212). And Brainard wrote, "We are all more or less unreasonable, and I only wonder that we are not all insane. All, including myself, are sullen, and at times very surly. If we are not mad, it should be a matter of surprise. I wonder if we will survive the horrors of this ice-prison" (p. 215).

At the start of the new year (1884) the incidents of food theft began to increase; on January 4 "Brainard reports that a hole has been cut through the canvas roof of the storehouse, and a small piece of bacon fished out. This bold attempt to steal our food gives me great uneasiness for the future, but the general sentiment is still strong and hearty in favor of equitable division among the strong, and such consideration toward the feeble as is possible" (p. 214). A few days later a hole was discovered in one of the barrels of bread, with several pounds taken out. Also, a piece of bacon was taken out while it was being cooked in a pot, and someone was scraping out and eating during the night the rancid oil used in an Eskimo lamp. A number of men began to show indications of scurvy. The mental condition of some of the men was causing concern: "Lieutenant Lockwood gave me such anxiety all night through," wrote Greely on January 13, 1884, "as at times he seemed to be decidedly out of his head. It appears that he has been saving up small amounts of each day's food; and from his own account, he ate to-day twenty-four ounces of solid food; an imprudence which has tended to break him down. He sees everything double and is very weak. He wanted rum frequently today, which I was unable to give him" (p. 221).

On January 18 the first man died, with the diagnosis of scurvy. The entry of January 22 records that 2 of the enlisted men were "impudent and insubordinate in their language." The discussions between the physician and other members of the party became at times very bitter and stopped barely short of violence. The physician was openly charged with selecting the heaviest dish from those issued by the cook. On February 13 Greely commented, "These continuous quarrels wear on me, for I never know how they will end, or what violence the wretched men may be tempted to commit, for in many ways death is preferable to life" (p. 297).

By the end of March, fishing for shrimps and shooting of game brought some relief, but the end was approaching with an ever more pressing certainty. On April 4 one of the Eskimos became delirious and died the next morning, followed on April 6 by another victim of starvation. Rice, one of the strong men of the party, perished on April 7 in an attempt to reach a cache of 100 lbs. of beef abandoned in November 1883. Lieutenant Lockwood was the next victim (April 9), and another man followed him on April 12. The shooting of a bear and a small seal staved off complete disaster for a matter of weeks. On April 19 the

physician was seen drinking the rum allowance of Elison, whose feet had been amputated some time previously; on another occasion he stole part of the sick man's bacon, hiding it in his sleeping bag. The death of Jens, an Eskimo, while on a hunt made the situation of the party even more precarious. On May 3 the last crumb of bread was gone. On May 19 the last alcohol was issued and one of the men died of starvation, followed on May 23 by other victims in rapid succession. Greely noted that even thinking was an effort and writing an exhausting activity. The men had no more strength to bury their dead and the entry of June 4 contains the following passage: "Our condition grows more horrible every day. No man knows when death is coming, and each has long since faced it unmoved. Each man who has died has passed into the preliminary stages of mental, but never violent, wandering without a suspicion that death marked him" (p. 315).

In the face of all the sufferings, Private Henry, who confessed stealing in the past, was detected stealing again and was shot on June 6, 1884. Schneider, near death himself, wrote after the death of the physician and another member of the expedition: "Lots of seal-skin and thongs were found on the doctor and Bender both, which showed how dishonest they were. Although Henry has told before his death that I had eaten a lot of seal-skin, yet, although I am a dying man, I deny the assertion; I only ate my own boots and a part of an old pair of pants. I feel myself going fast, but I wish it would go yet faster" (p. 322). By this time spring had opened up the meager vegetable larder of the far north — lichens, reindeer moss, rock tripe, and saxifrage, while the dry saxifrage was used for fuel. But death was reaping its harvest, one by one, inexorably. The entries in Greely's diary were getting shorter and shorter, the last being written on June 21.

When a relief expedition finally reached Greely's camp two days later, they found the men in a truly sad state (Schley, 1887, esp. pp. 44ff), only 7 of the party of 25 remaining alive: "Lieutenant Greely was in his sleeping bag. . . . Notwithstanding he had been told who we were he appeared dazed and asked if we were not Englishmen. . . . His mind wandered somewhat. His answers to questions appeared disconnected and at times incoherent; occasionally he would collect himself, apparently with some effort, but would soon indicate that his memory was indistinct. Pausing for a moment as if reflecting he would say, 'I am so glad to see you,' and almost immediately afterwards, 'Those lemons your wife so kindly put up for us,' etc. He had lain for weeks in his sleeping-bag, on account of gradually failing strength . . . all pain of hunger had ceased; his appearance was wild; his hair was long and matted; his face and hands were covered with a sooty, thick dirt; his form had wasted almost to a skeleton; his feet and joints were swollen; his eyes were sunken . . ." (*ibid.*, pp. 45-46). The condition of the other men was hardly less critical, and one of them died later, 3 days after the amputation of his frostbitten hands and feet. It was Schley's estimate that a 48-hour delay in reaching the surviving men would have been fatal to all who were still living.

As far as the question of cannibalism is concerned, Schley's description contains the following passage: "In preparing the bodies of the dead for transport

tation in alcohol to St. John's, Newfoundland, it was found that six of them had been cut and the fleshy parts removed to a greater or less extent with a view no doubt to use as shrimp bait. All other bodies were found intact" (p. 50).

Nansen's Sledge Journey in 1898

Nansen (1898), accompanied by one man, left the ship *Fram* on March 14, 1896, for a sledge journey to the north. Reaching the latitude of 86° N., at a longitude of 98°, they started on their march homeward on April 9 because the ice fields had become impassable and their food supplies were limited. After a 2 months' struggle, with only 3 dogs left, the food supplies started to run out. Nansen wrote: "We were hungry and toil-worn from morning to night and from night to morning, all five of us" (p. 111).

The response to food became strongly emotional. One day during the time they waited for the ice to open, Nansen wrote in a discouraged mood: ". . . it is a dull existence, no prospect of being able to get on, impassable packed ice in every direction, rapidly diminishing provisions, and now, too, nothing to be caught or shot." And what a contrast on the next day: "Saturday, June 22nd, Half-past nine a.m., after a good breakfast of seal's flesh, seal liver, blubber, and soup. Here I lie dreaming dreams of brightness; life is all sunshine again. What a little incident is necessary to change the whole aspect of affairs! Yesterday and the last few days were dull and gloomy; everything seemed hopeless, the ice impassable, no game to be found; and then comes the incident of a seal. . . . We have abundance of food and fuel for upwards of a month. . . . We have eaten our fill both at supper and breakfast, after being ravenous for many days. The future seems bright and certain, no clouds of darkness to be seen any longer" (p. 115).

After shooting another seal later, the men rejoiced to be able to eat as long as they found any room for the food. Seal's flesh, blubber, and blood were regarded as great delicacies. They found the blubber excellent both raw and fried, and used it also in place of butter. The meat was used for making soup and as steaks. Nansen's blood-pancakes fried in blubber were appreciated as a first-class meal. Later, bear meat was substituted for seal. Nansen commented that the exclusive meat and fat diet did not cause them any discomfort, that they did not get tired of it, and that they had no craving for farinaceous food. Having spent another winter in the wilds of the far north, the men finally reached Franz Josef Land the next summer.

De Long's Expedition

The expedition headed by G. W. De Long (E. De Long, 1897) had bad luck practically from the start and ended with the death of a large part of the crew. The ship left San Francisco in July 1879 and became locked in the ice the first winter of its journey. It had to be abandoned two years later when it was crushed by the ice in mid-June 1881. The crew marched over the frozen ocean, stopped on Bennet Island, and continued by boat until by the middle of September De Long with 13 of the men had reached the Lena Delta in northeastern Siberia. By this time the supplies of food were practically exhausted and the

men were suffering from overexertion and exposure. On September 21 they shot 2 reindeer, which staved off starvation for a time. On September 27, when provisions for only one more meal were left, another reindeer was shot; the men ate some 3 pounds of reindeer meat each before their desire for food was satisfied.

In the early days of October the daily ration consisted of half a pound of meat and tea. The exposure, the exhausting marches, the sleepless and cold nights were rapidly draining the physical energy. On October 3 the only remaining food was a bit of pemmican, some alcohol, and a half-starved dog. One man died on October 5. The last bit of meat was eaten on the morning of October 7; for dinner they had one ounce of alcohol in a pot of weak tea. Alcohol became the main source of calories. The physician of the expedition noted: "Alcohol proves of great advantage; keeps off craving for food, preventing gnawing at stomach, and has kept up the strength of the men" (*ibid.*, p. 792). Three ounces were served per day in hot water. But even the alcohol gave out on October 10. In his diary De Long noted the eating of deerskin scraps. There was nothing for supper but a spoonful of glycerin. "All hands weak and feeble, but cheerful. God help us." Because of a gale they were unable to proceed in their search for a settlement. On October 11 one spoonful of glycerin in hot water was their only food. On October 12 the diary contains the entry: "For dinner we tried a couple of handfuls of Artic willow in a pot of water and drank the infusion. Everybody getting weaker and weaker. Hardly strength to get firewood." The diary notes for the next 18 days are very brief. On October 15 the men had "willow tea and two old boots" for breakfast. Two days later one man died of exhaustion and starvation, on October 20 two other men passed away, and so it continued until October 30, the day of the last entry, written 140 days after the men left the unlucky *Jeannette*.

The other part of the crew was rescued, and later De Long's records were located. The language in which the hardships were described is reserved and objective and does not afford much insight into the psychological aspects of this tragedy of semi-starvation. Of special psychological interest is the facsimile of the last page of De Long's journal (pp. 798-99), which indicates that until the last day his handwriting remained essentially normal.

Scott's Expedition

Captain R. F. Scott, accompanied by 4 other members of his expedition, started the return trip from the South Pole on January 19, 1912 (Scott, 1923). The march was very difficult owing to blizzards and a rugged terrain. On January 25 Scott recorded signs of weakening in the party; only 2 men remained in full health. On January 28 Scott made the entry: "We are getting more hungry, no doubt the lunch meal is beginning to seem inadequate. We are pretty thin, especially Evans, but none of us are feeling worked out. I doubt if we could drag heavy loads, but we can keep going well with our light one. We talk of food a good deal more" (p. 439).

At times the situation appeared critical. The entry of February 12 is fairly representative: "... we went forward confident of covering the remaining distance, but by a fatal chance we kept too far to the left, and then we struck up-

hill and, tired and despondent, arrived in a horrid maze of crevasses and fissures. Divided councils caused our course to be erratic after this, and finally, at 9 p.m. we landed in the worst place of all. . . . After discussion we decided to camp, and here we are, after a very short supper and one meal only remaining in the food bag; the depot doubtful in locality. We *must* get there tomorrow. Meanwhile we are cheerful with an effort" (p. 493).

The food depot was found the next day, to the great relief of all. It provided supplies for the next few days, but the wear and tear of the trip was increasing. The physician and one of the two physically fit men suffered a severe attack of snow blindness. A frostbitten man developed huge blisters on the foot which delayed the progress of the march. The falls suffered during rough travel on the glacier probably resulted in brain injuries and, in combination with exposure and overexhaustion, led to the man's collapse, ending in coma and a quiet exitus on February 17. The party was making headway slowly, from depot to depot. Another man became seriously ill, and being unable to continue and unwilling to become a burden on the rest of the group, he left the tent bravely to meet his death in a -40° blizzard on March 17. But the days were numbered for all. The diary contains the following entry on March 20: "Sledge dreadfully heavy. We are $15\frac{1}{2}$ miles from the depot and ought to get there in three days. What progress! We have two days' food but barely a day's fuel. All our feet are getting bad."

On March 21 the expedition got within 11 miles of the food supplies but the severe blizzard made it impossible to leave their temporary camp site. The last entry, written 8 days later on March 29, ended thus: "Every day we have been ready to start for our depot *11 miles away*, but outside the door of the tent it remains a scene of whirling drift. I do not think we can hope for any better things now. We shall stick it out to the end, but we are getting weaker, of course, and the end can not be far. It seems a pity, but I do not think I can write more." The records were found later by a searching party.

Mikkelsen's Story

Mikkelsen's (1913) description of his semi-starvation experience in the Arctic is one of the richer sources of psychological observations. For the present purposes the major interest attaches to two men, Mikkelsen and Iversen, on the southward part of the journey. The finding of a food depot at a time when rations were getting very short for both men and dogs was a joyful event, and Iversen, who found the depot, came running back to the tent "breathless, and scarcely able to speak, but wild with delight." When the food was brought to the tent the men were at first overwhelmed by the variety from which to choose. "We are too hungry, however," wrote Mikkelsen, who was ill at that time (p. 228), "to waste much time in thinking it over. Iversen decided for lobscouse — half a tin — and as we imagine that oatmeal must be good for scurvy, I feast on porridge. The oatmeal is moldy, but never mind, a little mildew can't hurt very much, and we both agree that we never had a finer feast. We eat it slowly, a little at a time, tasting every spoonful, and when coffee is served after the banquet, we have nothing more to wish for."

The journey farther south was full of hardships on account of the terrain but for a time the food situation caused no concern. They developed a "knack" for finding even the well-hidden food depots. Mikkelsen commented: "... hunger has given us sort of a second sight, making it the easiest thing in the world to find anything in the shape of food" (p. 238). By the beginning of August 1910 their food supply was very low, and they were forced to subsist on little else than half a pound of pemmican per day. There was no problem of its becoming a monotonous diet: "... pemmican seems to us the most delicious dish that ever man tasted. It is our first thought when we wake in the morning, and it is a solemn moment when the pemmican boils. Spoonful by spoonful the precious stuff is shared out, lest one should get a drop more than the other, and even the well-scraped cooking pot is fairly divided, one licking the lid, and the other the pot itself, changing about at the next meal. This is eaten in the evening, twelve or fourteen hours later, and all day we long for the hours to pass, that we can get something to eat again" (p. 254).

The trip from Hovgaards Island to Danmarks Havn, reached on the eighteenth of September, was the most trying part of their travels. Chapter XI of Mikkelsen's book, appropriately entitled "Race against Hunger," is rich in details of psychological interest. The men arrived on Lambert's Land on August 15, exhausted with cold and hunger, with no food whatsoever. The countryside seemed promisingly rich in game. The description of the hunt is an excellent presentation of the behavior of a semi-starved man who has a prospect of satisfying his hunger: "We separate in order to cover as much ground as possible, Iversen going inland, while I hurry along the coast, with eyes and ears strained to catch the slightest sight or sound. But it is tiring work, even when every fibre in one's body is clamouring for food, and as time goes on, and watchfulness remains still unrewarded, the strained senses grow dull, and the unnatural energy which flared up at the sight of the goodly land dies down again and disappears. Utterly tired out, I tramp mechanically over the stony land, which now seems hopelessly desolate" (p. 260).

Fortunately, his comrade had better luck and met Mikkelsen with 12 ptarmigans in the bag: "... we wend contentedly homeward filled with one great thought of food. Time after time we stop to count them, and make sure we have not dropped one on the way. . . . The feast that followed I need not describe. Inside the tent we revelled on boiled ptarmigan, and outside the dogs devoured bones, entrails, feathers, everything; nothing was wasted" (p. 262). The birds did not last long, and the men were forced to kill the last 2 dogs. Against their better judgment they ate the dog's liver, which had a narcotic effect. Mikkelsen dozed off to a dream of food: "enormous quantities of food, huge smoking joints, mountains of bread and butter, with great green piles of vegetables and salad. But it is all moving, moving continually; shifting just out of reach. I run and run, it is always there, a little farther on; at last I fell and cut my head so that blood runs down my face. Then I woke, bathed in sweat, and with a frightful headache" (p. 270).

Food and rest became the focus of their thinking: "We have but one thought — beyond the eternal desire for food — to get on as fast as possible and make an

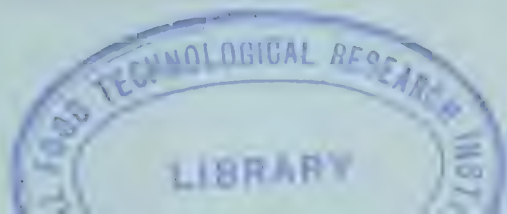
end to this eternal journeying" (p. 271). With only one ptarmigan between them and starvation, the men were overjoyed at finding a food depot on August 25. Mikkelsen noted, after the men had eaten their fill: "It is long since we have been able to talk of anything but food, and now that our hunger is for the moment appeased, it is quite a relief to talk of something else" (p. 279). The stock of provisions allowed the men to recover some of their strength. Rations had to be cut quite short again before they found another depot on September 4. The place of the next depot was found but the supplies had vanished. Eight pounds of food was woefully inadequate for covering the remaining 100 miles! And the next depots were empty as well. But the men toiled along.

"Our feet are very painful, the ankles being swollen and horribly tender, as a result of continually getting twisted among the rocks. As soon as we are fairly started, however, it is not so bad. The pangs of hunger are worse, increasing every minute, and causing us physical pain. For my own part, I can think of nothing but food. At first my thoughts dwell with fond recollections upon all sorts of dishes, but gradually they concentrate themselves upon sandwiches — Danish sandwiches, with no top slice, very different from the dull, dry things one gets in England. Otherwise I have for the last few days dreamed chiefly of enormous juicy steaks, as the most desirable human delight, but today it is sandwiches. Why I do not know, but so it is. In particular my fancy turns upon the many packets of delicious food which I have seen given away to beggars, and I grow quite furious at the thought of the contempt with which these gentlemen too often regard such gifts; treasures that I would give years of my life to buy. I remember the neat little packets of sandwiches from my schooldays, and gradually the thought takes possession of me to such a degree that at last I imagine that I am walking in the streets of Copenhagen, eagerly on the lookout for a packet of sandwiches. Suddenly I spy what I am seeking, a little white object lying a little to the right of me. I turn to pick it up before any one else can get it, but, as I stop, my foot strikes against a stone. The shock brings me back to stern reality, reminding me with painful distinctness that I am in Greenland, far away from Copenhagen and all its sandwiches. So great was the force of the hallucination, however, that I have actually turned out of my course, to Iversen's great astonishment, and at my foot lies a little white stone. I take it in my belt, and go on again, but the little packages still haunt me, and before long I catch myself running off once more after little white stones, quite certain this time that there is no mistake" (p. 303).

The last few days of the long weary march taxed all the strength the men had left, and they reached Danmarks Havn "staggering . . . at every step, dragging our feet like old men, and halting every now and again to rest" (p. 308). Although they were aware of the dangers of overeating, they both succumbed to the temptation to eat and eat, and suffered from severe gastrointestinal upsets for 3 or 4 days.

Behavior during Rehabilitation from Starvation

Reports on behavior under conditions of semi-starvation and famine leave much to be desired in completeness and in detail but information about the re-



habilitation phase and after is even more grossly inadequate. There is reason to believe that the psychic trauma may have more lasting effects than the purely physical effects of famine. But the typical account is reminiscent of the classical fairy tale: the food arrived, or the rains came, and they lived happily ever after.

The persistence of physical disorders in rehabilitation has been reported in some studies (e.g. Butler *et al.*, 1945; Mitchell and Black, 1946; Adamson *et al.*, 1947). In some cases these reflect behavioral abnormalities. For example, gastrointestinal disturbances in the early stages of rehabilitation are exceedingly common; they seem to be a direct result of overeating, which is often not controlled in spite of strenuous efforts to persuade these persons to moderation. Not infrequently relative obesity develops; the appetite, long unsatisfied, exerts an uncontrollable dominance.

It is difficult to decide whether some of the pathological phenomena in rehabilitation should be classified as physical or psychological in origin. For example, among Canadian troops repatriated after almost 4 years in a Japanese prison camp one rather common complaint was excessive sweating; in some cases this was said to interfere with the man's taking an ordinary job 6 to 12 months later. Was the sweating an expression of a lack of inclination to get back to ordinary work?

Even after the danger of starvation is clearly gone, the recently starved man tends to persist in his anxiety about food. After relief had come to the Belsen concentration camp in 1945 the inmates continued to steal, hide, and hoard food (Niremberski, 1946). As the nutritional rehabilitation progresses, the pattern of complaints changes. At Belsen the patients first complained without spirit, hopelessly, but as they grew better their complaints became fierce, bitter, and resentful. This is entirely in agreement with the pattern of the Minnesota Experiment.

Behavior and Complaints in Experimental Starvation and Rehabilitation

THE field reports presented in the preceding chapter described the complaints and behavior of men under conditions of starvation and famine in rather an unsystematic way and without any attempt to measure the magnitude of the changes or to indicate the frequency with which a qualitative characteristic was observed in a given population. Certain symptoms, such as weakness and depression, have been mentioned repeatedly in field reports. The field observations served as a guide for the observers in the Minnesota Experiment during their formal and informal contacts with the subjects. Also, the questionnaires designed to record the reactions of the subjects to the semi-starvation stress were based, in part, on an analysis of the older literature on famine and starvation.

This chapter presents the picture of changes in experimental semi-starvation reconstructed on the basis of clinical observations, descriptive material obtained from the subjects, and various questionnaires. Before the results of the Minnesota Experiment are reported, a brief review is given of the previous experimental studies in so far as they bear on the complaints and behavioral changes in starvation.

Work Preceding the Minnesota Experiment

Starvation experiments preceding the Minnesota Experiment have relatively little to offer from the point of view of emotional adjustment and behavior. The "fasting geniuses," some of whom had a preconceived idea of the beneficial effects of abstinence from food, could hardly be expected to communicate information that would have any degree of general validity. One of the more fruitful reports is that on Levanzin. In a total fast of 31 days (Benedict, 1915) the subject was cheerful during the first week. From then on he had periods of depression and irritability. He complained little, except about the monotony of the program, and he reported no hunger pangs. He developed a pronounced oversensitivity to cold. His movements became slower but he was able to walk without assistance throughout the fast, during which his weight decreased by 13.25 kg. (21.9 per cent of the original body weight). His intellectual capacity and expressive power as judged in conversation did not seem to be affected.

Some of the classical effects of prolonged semi-starvation were observed in the experiment carried out by Benedict *et al.* (1919). In this study the average body weight of the 12 young men decreased from 67.0 kg. (September 30, 1917) to a final level of 59.8 kg. (February 3, 1918), a decrement of 10.5 per cent of

the pre-experiment value; the minimum of 58.8 kg. was reached around the middle of December 1917. The subjects reported feelings of general weakness and tiredness. They complained of weakness in the knees, noticed particularly in climbing stairs. They had a poor tolerance to cold, asking permission at times to wear sweaters during athletic exercises and seeking to be close to the radiators whenever possible. They had to be relieved from some swimming exercises because the water in the pool seemed to them severely cold. Their endurance in active sports was reduced, and in calisthenics they could not keep up with the other men in the class. In the judgment of the athletics instructor they appeared to have poorer motor control and strength in the apparatus exercises.

The records of voluntary activity did not show any marked change during the period of undernutrition, but this may be explained by the arrangement in the Carnegie Experiment whereby the men tended to compensate for outside meals by extra exercise. Motion pictures of the men taken 2 days before the end of the experiment during gymnastic exercises, chinning, and diving from a springboard did not reveal any noticeable difference between the subjects and normally fed young men. The neuromuscular tests were largely negative, and at the end of the experiment the men felt that the maintenance of their weight at that level would not interfere with a normal life and work adjustment.

The "stress" of the experiment was most marked during the initial period of relatively rapid decrease in weight. The men became irritable and were easily annoyed. They found it somewhat difficult to concentrate on their studies and were bothered by persistent thoughts about food and eating. They felt they had to drive themselves harder to accomplish the same amount of work, but their ability for mental work was not affected adversely. Judged by the grades they received, their college work was not inferior either to that of their classmates or to their own previous standards. A marked reduction in the sex drive was observed throughout the period of reduced diet. Attraction to the opposite sex was much lessened. Sex dreams were not recalled, and nocturnal emissions and erections became less frequent. This trend was reversed when the men returned to a normal, unlimited diet; normal sex interests reappeared, manifesting themselves in the desire to associate with women and in finding female companions attractive.

Methods — Minnesota Experiment

Complaints of the Minnesota subjects were recorded systematically in regularly scheduled interviews, conducted by 3 staff psychologists, and by means of standardized forms. In the self-rating questionnaire each subject rated a series of items, such as irritability or moodiness, on a 6-point scale extending from 0 (normal or absent) to 5 (extreme). In the complaint inventory no judgment was required about the intensity of a complaint; the subject merely noted either the presence or the absence of a particular symptom. This complaint inventory contained 53 items bearing on the various aspects of the physiological and the psychological status of the respondent. Typical questions were: "Do you have many headaches?" "Are you able to work as well as ever?" "Do you find that you do not particularly desire to mix socially with people?" The inventory required only the yes or no type of answer. In some items "yes" was the "unde-

sirable" response, in others "no." The individual score was the number of undesirable responses recorded for a subject. The average of these individual scores gives an indication of the over-all change in the adjustment of the group. The self-rating questionnaire and the complaint inventory cover essentially the same areas.

In addition, each man was rated as to the over-all deviation from his pre-starvation mental and physical status by a group of his close acquaintances. The number of men who served as raters for a given subject varied from 4 to 27; the raters remained the same throughout the experiment. The rating scale extended from 0 (no deterioration) to 5 (extreme deterioration).

The staff had ample opportunity for firsthand observations of the behavior of the subjects and for clinical impressions during frequent formal and informal contacts with them. The men were observed under such varied conditions as individual and group testing, personal interviews, clerical and technical laboratory work, and participation in group meetings, as well as at times when the staff members ate with them.

Diaries kept by the subjects provided an additional source of descriptive information. This material was used primarily in the case studies presented in Chapter 41.

General Results of Questionnaires

Results of the self-ratings are given in Table 360. The ratings for the control period were made after 3 months of standardization, and the values were essentially zero (normal) for all the items. The items in which there was the greatest amount of deterioration during starvation were "tiredness" (mean score at S24, +3.5), "appetite" (+3.1), "muscle soreness" (+2.1), "irritability," "apathy," and "hunger pain" (+1.8), and "ambition," "self-discipline," and "concentration" (-1.8). The men liked the semi-starvation food better than the more varied diet served during standardization, but the increased "palatability of the food" can hardly be regarded as a symptom of "deterioration."

Some symptoms — "moodiness," "depression," and lack of "self-discipline," for example — became more severe as semi-starvation progressed. On the other hand, in "hunger pain" there was little change after S12. In some items ("dizziness," "nausea," "salt craving") the trend was actually reversed, although in these cases the differences between the mid-starvation (S12) and the final semi-starvation (S24) ratings were small.

After 12 weeks of rehabilitation there was a marked improvement in all symptoms, the continued desire for more food and the feeling of tiredness being the most prominent semi-starvation residues. By R20 the values had returned to near-normal levels, and by R33 the rehabilitation, as judged on the basis of self-ratings, was essentially complete.

A similar rating system was used for 3 "drives" which were considered of special importance in a starvation-rehabilitation experiment: concern with food, sex, and need for activity. The course of the changes in the ratings is charted in Figure 112. The rise in the curve representing the "food drive" corresponds to the elevated ratings for "appetite" and "hunger pains." There is a decrease in the

TABLE 360

SELF-RATINGS. An increase in the particular symptom was rated on a scale from More = 1 to Extremely More = 5; a decrease was rated on a scale from Less = -1 to Extremely Less = -5; absence of a symptom or its presence in a "normal" amount was assigned a value of zero. At 12 and 24 weeks of semi-starvation (S12 and S24) and at 12 weeks of rehabilitation (R12) the values are averages for 32 subjects; at 20 and 33 weeks of rehabilitation (R20 and R33) the averages are for 30 and 20 subjects, respectively. (Minnesota Experiment.)

Complaints	S12	S24	R12	R20	R33
Hunger pain	1.69	1.75	.38	0	0
"Appetite"	2.72	3.06	1.56	.37	.20
Palatability of food ...	2.31	2.16	.88	.30	-.05
Salt craving97	.84	.41		0
Nausea39	.32	.16	.03	0
Dizziness	1.53	1.34	.19	0	.05
Fainting31	.34	0	0	0
Tiredness	2.69	3.47	1.19	.47	.30
Muscle soreness	1.32	2.13	.55	.28	
Muscle cramps55	1.39	.32	.07	
Depression69	1.38	.34	.10	0
Moodiness84	1.50	.38	0	0
Irritability	1.31	1.81	.56	-.03	-.15
Apprehension34	.41	.25	-.03	0
Apathy	1.09	1.81	.41	.03	.10
Sensitivity to noise ..	1.31	1.81	.38	0	
Ambition	-1.19	-1.75	-.50	0	0
Self-discipline	-.56	-1.72	-.41	0	-.15
Mental alertness	-.84	-1.53	-.31	.07	-.05
Concentration	-.91	-1.66	-.62	.07	.05
Comprehension	-.44	-1.03	-.38	.07	0

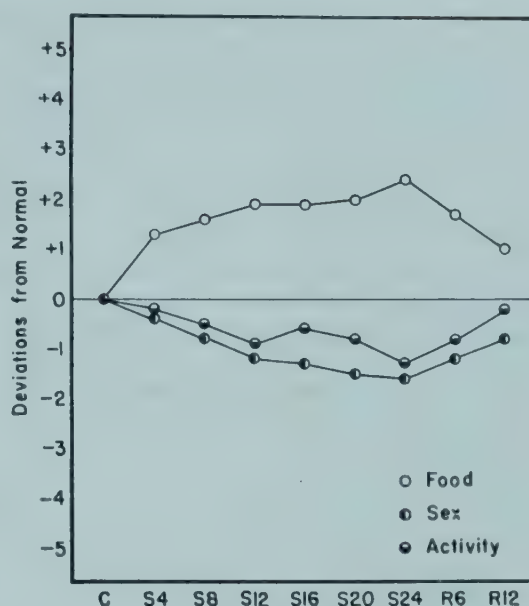
TABLE 361

SELF-RATINGS OF IMPROVEMENT IN VARIOUS SEMI-STARVATION SYMPTOMS. The values represent the average percentage estimates of the degree of recovery during the first phase of rehabilitation (at R6 and R12). Full recovery = 100 per cent.
Number of subjects = 32. (Minnesota Experiment.)

Physical Symptoms	No Deterioration*	Recovery (%)		Psychological Symptoms	No Deterioration*	Recovery (%)	
		R6	R12			R6	R12
Increased sensitivity to cold (1)		46	77	Irritability	(1)	41	70
Increased sensitivity to noise (9)		41	68	Nervousness	(5)	45	74
General slowing down	(0)	41	68	Indecisiveness	(3)	48	72
Visual difficulties .. (20)		56	75	Preoccupation with thought of food ..	(0)	33	61
Loss of sex drive ... (2)		30	57	Loss of sociability ..	(1)	40	66
Hunger	(0)	38	65	Apathy, depression .	(2)	51	79
Tiredness	(0)	30	54	Lack of energy	(0)	39	64
Weakness	(0)	33	61	Fluctuations in mood	(0)	45	70
Dizziness	(4)	60	89	Flightiness	(4)	39	68

* These numbers in parentheses indicate the number of men who noted during semi-starvation no change in the particular item and who consequently could not "improve" during rehabilitation. Thus the percentage recoveries are based on those individuals who did suffer a semi-starvation deterioration with respect to a given item.

FIGURE 112. AVERAGE RATINGS OF THE FOOD, SEX, AND ACTIVITY DRIVES for 32 subjects at different stages of the Minnesota Experiment.



“activity drive” and a more marked change in the “sex drive.” The *course* of the curves conforms closely to the clinical impressions; the *magnitude* of the deviations from the normal appears to have been underestimated by the subjects, especially for the sex drive.

At R6 and R12 the men were asked to estimate the percentage of recovery in the various physiological and psychological areas in which they had exhibited marked changes during semi-starvation. Essentially this amounted to a rating of the amount of recovery on a 10-point scale. We recognize the limitations of such “quantitative” estimates, but they are helpful in indicating the rate of recovery as experienced by the subjects. In the Minnesota Experiment these estimates may have greater validity than is usually the case because the subjects were highly intelligent, were trained in self-evaluation, and were not biased, their only concern being to bring out the most accurate picture of events. The data in Table 361 indicate that improvements in dizziness and in apathy were most rapid and most complete, while tiredness, decrease of the sex drive, and weakness were slow to improve. Even at R12 the men judged themselves to be far from the pre-starvation normal in these characteristics. The records for 12 men who rated their degree of rehabilitation at R20 indicated a nearly complete return to normality. The data obtained by this technique correspond closely with the picture of rehabilitation derived from the self-ratings presented in detail in Table 360.

The average values of the intersubject (man-by-man) ratings for the group were 1.6 at S12 and 2.3 at S24, decreasing to 1.4 at R6 and 0.7 at R12 (0 = no deterioration; 5 = extreme deterioration).

The complaint inventory was administered at approximately monthly intervals throughout the experiment. The scores were based on the total number of complaints or symptoms. The average number of undesirable responses per man rose from 6.6 in the control period to 20.5 at the end of semi-starvation. At R12 it had decreased to 11.3. For the group of 20 men for whom complaint inventory scores were obtained in March 1946, the average scores were 6.0 at C, 19.4 at S24, 9.8 at R12, 4.7 at R20, and 2.6 at R33. It may be noted that the undesirable answers were somewhat more numerous at C than at R33. At the time the control data were obtained the subjects had already been under the restrictions and limitations imposed by the standardization and testing regimen for almost 3 months. During this period some subjects were required to lose weight in order to ap-

TABLE 362

PERCENTAGE FREQUENCY OF UNDESIRABLE RESPONSES IN THE COMPLAINT QUESTIONNAIRE. For each item the "undesirable" answer is indicated in brackets. Thirty-two men completed the questionnaire at each period except the last (R33), when only 20 men were involved. C = control; S24 = 24 weeks of semi-starvation; R12, R20, and R33 = 12, 20, and 33 weeks of rehabilitation. (Minnesota Experiment.)

	C	S24	R12	R20	R33
Hunger pain					
Are you troubled by pains in your stomach or gut? [YES]	(3)	34	6	6	5
Have you experienced any physical discomfort (including pains) due to hunger? [YES]	(19)	62	19	0	0
"Appetite"					
Has there been a decrease in your craving for food? [NO]	(69)	72	38	6	5
Do you feel hungry almost all the time? [YES]	(12)	59	25	9	5
Palatability of food					
Does the food you get taste good to you? [NO] ...	0	0	9	3	5
Are you indifferent about the taste of food as compared with the quantity offered to you? [YES]	19	47	34	6	10
Nausea					
Are you troubled by attacks of nausea and vomiting? [YES]	0	3	3	0	0
Fainting					
Have you had blank spells in which your activities were interrupted and you did not know what was going on? [YES]	0	9	0	0	0
Coordination					
Have you noticed that your hands shake or are more awkward and clumsy than they used to be? [YES]	0	41	3	6	0
Have you felt any unsteadiness and uncertainty of your footing in walking? [YES]	0	94	28	3	0
Ability to concentrate					
Are you finding it hard to keep your mind on one job? [YES]	12	66	25	0	0
Sometimes when you try to read or study are you so restless that you cannot sit still for any length of time? [YES]	22	41	19	9	5
Mental alertness					
Is your judgment as good as it was before participating in this experiment? [NO]	0	41	38	0	0
Can you understand what you read as well as you could before the experiment? [NO]	0	41	28	6	0
Apprehension					
Do you frequently find yourself worrying about something? [YES]	9	12	28	12	0
Are you generally uneasy and uncomfortable about what might happen to you in the future? [YES]	3	6	3	9	0
Do you frequently feel anxiety about something or someone? [YES]	6	16	16	6	0
Apathy					
Does your daily life contain many things which keep you interested? [NO]	0	9	9	9	0
Do you frequently feel that you don't care what happens to you? [YES]	6	9	3	0	0

TABLE 362 *continued*

	C	S24	R12	R20	R33
Apathy (continued)					
Even though things are going all right for you, do you often feel that you are indifferent and unconcerned? [YES]	16	34	12	9	10
Ambition					
Do you find it easier these days to give up hope that you will amount to something? [YES]	3	19	3	0	0
Are you about as able to work as ever? [NO]	0	97	78	34	10
Have you felt more like giving up when things go wrong than before? [YES]	3	78	28	6	5
Self-discipline					
Are you almost always able to carry through the ac- tivities you have planned for yourself? [NO]	25	59	38	9	10
Does it seem that your plans are often so full of difficulties that you have to change them or give them up? [YES]	0	47	12	6	0
Tiredness					
Do you find yourself often sleepy and/or tired during the day? [YES]	41	88	50	38	35
Do you frequently have the sensation of being weak all over? [YES]	3	81	19	3	0
Do you tire quickly? [YES]	16	97	75	44	15
Neurological symptoms					
Do parts of your body often have feelings like burning, tingling, crawling, or "going to sleep"? [YES]	0	59	25	0	0
Does it seem that there is a lump in your throat much of the time? [YES]	0	6	0	0	0
Do you have many pains? [YES]	3	3	0	0	0
Do you have many headaches? [YES]	0	0	9	0	0
Depression					
Do you have spells of the "blues"? [YES]	16	31	22	12	5
Are there more times when you feel downhearted than before? [YES]	3	62	22	12	0
Are you fairly often disappointed in people or in the outcome of situations in which you have had some interest? [YES]	31	41	22	16	10
Moodiness					
Do you find that you are sometimes happy and sometimes sad for no particular reason? [YES] ...	25	44	16	19	10
Irritability					
Do you find yourself impatient when people question you or otherwise interrupt you when are busy? [YES]	6	44	22	12	5
Does "guinea pig" life seem to be a strain for you much of the time? [YES]	0	62	12	(3)	(0)
Do your feelings seem to be more easily hurt than formerly? [YES]	3	22	9	6	0
Do you seem to be more susceptible to feeling pain than you were? [YES]	0	9	6	0	0
Memory					
Does your memory seem to be all right? [NO]	3	28	9	3	0
Concern about health					
Are you worried about catching diseases or becoming ill? [YES]	0	0	0	0	0
Are you in just as good physical health as most of the other guinea pigs? [NO]	3	6	0	6	0

TABLE 362 *continued*

	C	S24	R12	R20	R33
Sociability					
Do you find that you do not particularly desire to mix socially with people? [YES]	9	59	38	12	10
In social conversations are you usually a listener rather than a talker? [YES]	47	72	41	34	20
Do you prefer to be left alone and to be by yourself more than formerly? [YES]	6	50	19	3	5
Are you frequently bored with people? [YES]	6	62	22	9	10
Group status					
Do you sometimes strongly wish that you had not volunteered to be a guinea pig in this experiment? [YES]	0	12	0	0	0
Do you think that you are as much liked by the other guinea pigs as most of the fellows? [NO]	6	9	6	(3)	(0)
Do you wish that you were as happy as the other guinea pigs in general seem to be? [YES]	19	19	9	(22)	(15)
Average number of "undesirable" responses per subject	6.6	20.5	11.3	6.6	2.6

proach their "standard" weight more closely, and the testing requirements were arduous and frequently inconvenienced the subjects. Consequently, it is to be expected that post-experimental reports might contain fewer "complaints."

In addition to the over-all scores (the average frequency of undesirable responses *per subject*), the percentage frequencies of individuals answering different *items* in the undesirable direction at the various stages of the experiment are illuminating. These frequencies are tabulated in Table 362, where the items are grouped to conform to the order of the self-ratings (in the actual inventory the items were presented to the subjects in random order, without category headings). An attempt was made to cover most of the points by more than one question, except for such unequivocal items as nausea and fainting.

The sensitivity of the single items to the semi-starvation stress varied widely. Unsteadiness and uncertainty of footing in walking, inability to do hard physical work, and rapid onset of tiredness were universal at S24. On the other hand, not a single subject complained that the food he got did not taste good, worried about catching a disease, or had many headaches.

For some items the data for the control period were not valid "normal" base lines. This is particularly true of the first two categories, hunger pain and appetite. As noted above, some of the subjects were already on a moderately reduced food intake during this period and could truthfully answer that they felt hungry almost all the time. The item concerning the decrease in the craving for food is of importance for characterizing the rehabilitation period, and it is the semi-starvation (S24) frequency that must serve as the basis of comparison.

In the following six sections of this chapter the semi-starvation changes in various aspects of the subjects' behavior are discussed in greater detail with reference to other sources of information, including interviews and, to a limited extent, the subjects' diaries (see Franklin *et al.*, 1948). We shall return to the data obtained by standardized questionnaires and rating schedules in discussing

the effect of dietary factors on the course of the first phase of nutritional rehabilitation (R1 to R12).

Physical Changes

Striking changes took place during the course of semi-starvation in the physical appearance of the subjects; these are described in detail in Chapter 7. Many of these changes, such as protruding ribs or "baggy" knees, were of psychological importance since they made the subjects more acutely aware of their semi-starvation deterioration.

Both the face and the body showed marked emaciation. The gradual wasting of muscle and subcutaneous adipose tissues made sitting on hard surfaces uncomfortable. Shoes were too large and clothes were loose and poorly fitting. By the twelfth week of semi-starvation the edema became common and was especially noticeable in the knees, ankles, and face. The subjects reported that their nails grew more slowly and that their hair was falling out in large amounts. Shaving was necessary less frequently. The men noted, particularly in shaving, that cuts and wounds bled less than normally and were slower to heal. Physical ability to laugh heartily, sneeze, or blush was reduced or absent during the later stages of semi-starvation. Muscle cramps and particularly muscle soreness were frequently reported. The jarring of knee joints, especially when walking on hard pavements, was an annoyance to some. Pigmentation, thinning, and roughening of the skin occurred. Changes in the sensitivity of the skin, paresthetic and hypesthetic in character, were observed in only three cases, but there were many complaints that the extremities "went to sleep."

Tolerance to heat was increased; for example, subjects could hold hot plates without discomfort. They asked that their food, coffee, and tea be served unusually hot. Conversely, cold temperatures were poorly tolerated. Complaints of being cold or of having cold hands and feet were frequent and persistent. In hot summer weather many of the subjects slept under heavy blankets and wore extra clothing during the day. Vertigo, giddiness, and momentary blackouts were experienced on rising from lying or sitting positions by almost all the subjects during the first months and by some subjects throughout the semi-starvation period. Actual fainting occurred spontaneously on only one occasion. A few other incidents of "fainting" were reported, but the loss of consciousness was either very brief or incomplete.

Objective tests revealed no impairment of visual acuity but many subjects complained of transient visual disturbances such as inability to focus, eye-aches, and "spots" before their eyes. Standard measurements of hearing showed a slight but consistent increase in auditory acuity during the period of semi-starvation. It is difficult to determine whether the frequent complaints that ordinary sounds and noises were disturbing and annoying had a direct physiological basis in the "improved" auditory sensitivity or were primarily signs of an increased irritability. Sensations of ringing in the head were reported. Except for hunger pains and some decrease in the frequency of bowel movements, gastrointestinal symptoms were rare.

The marked decreases in pulse rate and basal metabolism may be regarded

as critical indicators of a lowering of speed in the automatic functions of the body. Other aspects of the machinery of the body exhibited a similar tendency. The responses of the tendon reflexes became more sluggish. Voluntary movements also became noticeably slower, and energy output was in general markedly reduced. However, over-all energy expenditure was maintained on a relatively high level by scheduled physical activity (walking to and from the mess hall, hiking about 20 miles per week, etc.). The attitude of the men toward physical exertion was ambivalent. It made them tired and as a rule was avoided. On the other hand, some men exercised deliberately at times. Some of them attempted to lose weight by driving themselves through periods of excessive expenditure of energy in order either to obtain increased bread rations (when weight loss exceeded the prescribed rate) or to avoid reduction in rations (when weight loss lagged). Most of the men felt weak and tired easily. They moved cautiously, climbing stairs one step at a time. Coordination was affected; the men sometimes tripped over curbstones and bumped into objects which they intended to side-step.

The marked reduction in strength and endurance was paralleled by a general curtailment of self-initiated, spontaneous activities. As starvation progressed, fewer and fewer things could stimulate the men to overt action. They described their increasing weakness, loss of ambition, narrowing of interests, depression, irritability, and loss of libido as a pattern characteristic of "growing old." The subjects were not acutely apprehensive about their health. None evidenced anxiety over the possibility of permanent effects of the undernutrition. They were confident that they would receive prompt and adequate care if they became ill and also that they would be removed from the experiment if it was thought likely that serious consequences would develop from their continued participation in it.

During rehabilitation the recovery from dizziness, apathy, and lethargy was most rapid. Tiredness, loss of sex drive, and weakness were slow to improve. Although visible edema tended to disappear, in some men there was little change or even an increase in edema. Cramps, vague aches and pains, and paresthesias were unrelieved for some time. Some of the men had new complaints such as flatus, distention, belching, and stomach-ache. Those subjects who gained the most weight became concerned about their increasing sluggishness, general flabbiness, and the tendency of fat to accumulate in the abdomen and buttocks. At the end of 3 months of rehabilitation (R12), even in those subjects who were maintained on the highest caloric intake the over-all physical condition was considerably inferior to the pre-starvation status. Later reports from the subjects indicated that it was not until after an additional 3 months of "normal" living and supernormal eating that their physical capacity approached pre-experimental levels.

Hunger

The qualitative aspects of the complex experience which one normally has some hours after a meal were described in detail by Stavel (1936), who reviewed the older literature on the topic. The successive phases are characterized by: (1) diffuse restlessness; (2) sensation of tension in the pharynx and the

upper part of the esophagus; (3) emptiness, without any sharp localization in the epigastric area; and (4) acute experience of hunger, characterized by unpleasant, aching, gnawing sensations ("hunger pangs"). The impulse to quiet these tensions by eating grows in intensity and may be associated with anticipation of the tactile, thermal, and kinesthetic sensations customarily connected with eating.

No attempt was made to follow the fine nuances of the qualitative phenomena of hunger in the Minnesota subjects or the changes in these during successive phases of the experiment, since this would have required considerable training in introspection. In speaking of "hunger" the subjects referred to sensations vaguely localized in the abdominal region which varied from mild discomfort to intense pain. In total starvation the sensation of hunger disappears in a matter of days. In semi-starvation there was no diminution of hunger. While some subjects suffered relatively little distress from hunger, others complained of being hungry all the time.

The menu containing the most bulk was the most popular of the three to which the Minnesota subjects were limited. The desire for variety in diet became very strong at times but was always subordinate to the stronger craving for greater quantity. The men not only drank considerable amounts of fluid in the form of coffee and very "soupy" soup in an effort to "fill up" but they demanded that the food and beverages be hot. It is of interest that Aristotle reported that warm food was more effective than cold food in counteracting the sensation of hunger.

The theories of hunger were summarized by Cannon (1929), who was concerned with hunger as a sensation. The older theories considered hunger a "general sensation" based on the need of the body for food (Magendie, 1817, cited by Boring, 1942, p. 571). Cannon favored a theory accounting for the origin of the hunger sensation by the strong contractions of the muscular wall of the stomach; the hypothesis was based on observations of the association between the periodically recurring hunger pangs and the sounds of moving air in the stomach region. Using a technique developed for experiments on dogs by Bol-direff (1905), one of Pavlov's pupils, Cannon made observations in 1911 on a trained human subject who was accustomed to the presence of a gastric balloon and to a tube in the esophagus leading from the balloon to the registering mechanism; the subject depressed a key when a sensation of hunger was present. The reported hunger pangs coincided with marked decreases in the volume of the gastric balloon that indicated increases in the degree of contraction of the stomach (Cannon and Washburn, 1912). These observations were confirmed and extended by Carlson (1912, 1916) and Wada (1922).

Cannon's theory of the local initiation of hunger sensations fits some of the facts observed in the Minnesota Experiment. The men attempted to keep the stomach full by consuming large amounts of liquids and felt they were less hungry as a result. It is known that under conditions of natural starvation people fill their stomach with all kinds of materials, even clay (cf. Cannon, 1929, p. 436). The relief of hunger pain by such means is to be explained by the mechanically induced inhibition of gastric movements. Among factors that tend to

inhibit hunger contractions are chewing and smoking (Carlson, 1916); both were indulged in by a large number of the Minnesota subjects.

We did not measure stomach movements. However, the observations made during semi-starvation and, even more importantly, during rehabilitation indicate that the textbook concept of hunger as a gastric sensation due to the strong peristaltic contractions *arising in the empty stomach* (Best and Taylor, 1945, p. 519) is not tenable. In the rehabilitation period our subjects frequently reported that they were hungry immediately after a large meal. The hunger pangs present under these conditions *may* have been due to an exaggerated tonus of the stomach, of course, but it is certain that they were not caused by the emptiness of the stomach. Janowitz and Ivy (1949) believe that "the gastric 'hunger' contractions and the associated epigastric pang are but one and a dispensable component of the entire complex of hunger sensations" (see also Grossman and Stein, 1948).

The reasons for an increase of the feelings of hunger in semi-starvation are not clear. Our evidence is mostly on the negative side, eliminating factors that could be considered causally related to the phenomenon of hunger. It appears that the increase in hunger pangs, present in the large majority of the subjects, could not have been due to an over-all hyperactivity of the stomach; the increase in the time of emptying of the stomach, discussed in detail in Chapter 26, indicates that gastric motility actually decreased. Another objective indication of decreased tonus was a pronounced visceroptosis, a relaxation of the gastrointestinal musculature expressed in the lowering (ptosis) of the stomach into the abdominal cavity as shown by the X-ray pictures. There was no question of hyperacidity causing irritation of the stomach walls and in that way arousing gastric sensations which might be interpreted as hunger.

The blood sugar level was lowered in semi-starvation, both at rest and, more markedly, at work. But this factor can hardly account for the over-all increase of hunger, at least as far as the mechanism of gastric "hunger contractions" is concerned. Quigley and Halloran (1932) found in dogs that intravenous injections of glucose into the empty stomach or placing sugar in the duodenum caused inhibition of gastric motility in 3 to 5 minutes; this was interpreted not as a result of hyperglycemia but as a reflex from the duodenum. Mulinos (1933) presented data indicating that in dogs the motility of the empty stomach is not related to physiological (or spontaneous) variations in the blood sugar and is not influenced by raising the blood sugar concentration with intravenous injections of glucose. On the other hand, insulin hypoglycemia was accompanied by increased gastric motility, and this could be depressed by oral or intravenous administration of glucose.

Similar observations were made on man by Janowitz and Ivy (1949) except that hunger sensations, not gastric contractions, were used as the criterion. The fluctuations in blood sugar levels which occurred in fasting subjects, with initial blood sugar levels ranging from 71 to 89 mg. per 100 cc. of blood, were small (4 to 9 mg. per cent) and were in no way related to the hunger sensations. Intravenous injection of glucose, resulting in hyperglycemia (maximum levels from 143 to 210 mg. per cent), had no detectable effect on the spontaneously

occurring hunger sensations. Intravenous injection of insulin induced hunger in all subjects. On the average, the hunger sensations were reported 27 minutes after the lowest blood sugar level was reached. At this time the blood sugar concentration was 42 mg. per cent. The hunger sensations subsided when the concentration rose to 61 mg. per cent, a level which was still far below the fasting level.

Morgan (1943, pp. 665ff) pointed out with much justification that the *sensation* of hunger (and appetite, considered by Cannon to be a learned craving for appealing food) covers only a small and, as a result of the studies in animal behavior, a decreasingly important aspect of the problem of food intake. Boring (1942, pp. 551-58) followed historically the transition from the concept of hunger as a conscious experience to the concern with hunger as a drive for food. It would simplify matters if a term other than *hunger* were used for this drive. The presence of this food drive may be inferred from the behavior of animals and men from whom the stomach has been removed and who, consequently, cannot suffer from hunger pangs of gastric origin; rats with experimentally removed stomachs exhibit behavior indicative of the strength of the hunger drive which is not different from that of the control animals, except that the animals without stomachs eat more often (Tsang, cited by Morgan, 1943, p. 447). Consequently, the food drive is a more fundamental factor in the seeking and eating of food than the hunger sensations related to gastric motility. Even though the wide variations in blood sugar level were not found to be correlated with hunger sensations and at present no good ideas have even been suggested about the nature of a "hunger hormone," it is certain that the factors stimulating the ebb and flow of food intake must be related to the changing metabolic state of the organism.

We attempted to obtain information on the strength of the hunger drive by means of self-ratings. This is not a satisfactory procedure because it is impossible to differentiate sharply enough the behavioral manifestations of the food drive from hunger (a dull, aching, gnawing sensation localized roughly in the region of the stomach) and from appetite (a craving for food, not referred to any special region of the body). Because our experimental situation was so structured that the strength of the drive could not be tested, except in an all-or-none manner, the ratings provide little genuine information on hunger as a motive. The manifestations on which the ratings were based are related primarily to the degree of the felt need for food, the preoccupation with thoughts of food, and the temptations to eat off diet.

It should be stressed that the discomfort caused by hunger pain is by no means the most important change resulting from semi-starvation. In men who were unable to adhere to the diet, as well as in those subjects who exhibited a severe psychological deterioration (their case histories are presented in detail in Chapter 41), the "hunger pangs" did not seem to be present in any manifestly exaggerated degree.

Adherence to Diet

The subjects were aware of the scientific and practical importance of the experiment and each man felt that his participation in the program would result

in a definite contribution to the body of knowledge in an important field. They were convinced that the findings would aid in the relief and rehabilitation of the starving peoples of the world. Many of the subjects hoped to be able to go abroad as members of nutritional relief teams, and they felt they would be much better able to understand and to minister to famine sufferers by virtue of their firsthand starvation experiences. These factors provided strong incentives to adhere strictly to the diet. Although some temptation to break the diet was present at times, the voluntary commitment to complete the experiment as planned was, with few exceptions, greater than the tantalizing craving for food. This is attested by attainment of the required weight loss. Some men maintained that eating other food than that provided by the diet simply never occurred to them. Direct or indirect evidence of nonadherence to the diet was obtained for 4 out of the 36 subjects; the case histories of these 4 are given in some detail in Chapter 41.

In the beginning of the semi-starvation period the subjects were allowed to go about alone, but later they were required to be accompanied always by a "buddy." If invited out, the subject could take along a part of his own food (probably a cold macaroni sandwich or a plain slice of bread). Under such conditions the men disclaimed any conscious temptations and were annoyed when hosts or friends tried to persuade them to take just a little extra food. The use of subjects in the Laboratory on jobs involving the handling of food was discontinued at their request because the temptations under these circumstances were too great; they found themselves inadvertently licking gravy off their fingers or picking up crumbs.

Eating Habits

Anticipation of eating heightened the craving for food. Consequently the men dreaded waiting in line while their meals were being measured and weighed, and each man defensively guarded his place in line. They tended to become irritated when the serving was slow or when those who served the food gave any evidence of not taking their business "seriously." The men all considered it extremely important that the food be served very hot. It was as though the starving individual "borrowed" heat from the food ingested as a means of conserving energy.

The subjects exhibited a possessive attitude toward their food. At the table some hovered low over their trays with their arms placed as if to protect their ration. For the most part, they ate silently and deliberately and gave total attention to the food and its consumption. Many ingenious devices were used to make the food seem to go farther and to provide the illusion of variety. As the starvation progressed, the number of men who toyed with their food increased. They made what under normal conditions would be weird and distasteful concoctions. There was a marked increase in the use of spices and salt. Park (1918), a Canadian who became a prisoner of war and was employed from 1916 to 1918 as a medical officer in the concentration camp of Minden, in Westphalia, also noted that many of the men who were receiving insufficient rations, given almost entirely in the form of soup, used large quantities of salt. Toying with food was mentioned by Friedrich (1950), a German student who became a Russian pris-

oner of war. The undernourished patients in the hospital, to which he was sent because of his extreme weakness, played with their food for hours. Those who ate in the common dining room smuggled out bits of food and consumed them on their bunks in a long-drawn-out ritual. Some saved their bread during the whole week in order on Sunday to enjoy to the fullest a private eating ceremony, with a "table spread" on their bunk and fancy "silverware."

The Minnesota subjects were often caught between conflicting desires to gulp their food down ravenously and to consume it slowly so that the taste and odor of each morsel would be fully appreciated. Toward the end of starvation some of the men would dawdle for almost two hours over a meal which previously they would have consumed in a matter of minutes. To increase the pleasure of eating, they did much planning as to how they would handle their day's allotment of food. Since the subjects went to the dining room only twice a day during the starvation phase of the experiment, many of them saved out part of their meals for later consumption. All food was consumed to the last crumb. Although most of the men were well educated and refined, almost all of them routinely licked their dishes in order to obtain every vestige of food. They quickly became intolerant of food waste and were visibly upset when they noticed non-subjects in the same dining room discarding food. There were bitter comments that any spoilage or wastage of food was prodigal and criminal in a starving world.

Individual food dislikes for such diet items as rutabagas and fish disappeared in the early part of the semi-starvation period, confirming the age-old saying that "A hungry stomach rarely scorns common foods" (Horace's "Jejunus raro stomachus vulgaria temnit"). Although the subjects were restricted to 3 rather monotonous menus, the taste appeal of the diet increased rather than diminished throughout the 6 months of semi-starvation.

Preoccupation with Food

Preoccupation with thoughts of food is mentioned repeatedly in the field reports from semi-starvation areas. Characteristically, in a hospital for Germans who became Russian prisoners of war, eating (and bowel movements), not sex, was the principal topic of conversation, and heated arguments revolved around recipes (Friedrich, 1950).

Food in all its ramifications became the principal topic of conversation, reading, and daydreams for almost all Minnesota subjects. When they read books or attended movies, they were much impressed by the frequency with which food and eating were mentioned. Cookbooks, menus, and information bulletins on food production became intensely interesting to many of the men who previously had had little or no interest in dietetics or agriculture.

In order to obtain a more quantitative evaluation of some aspects of the concern about food, toward the end of semi-starvation the men were asked two questions: (1) Have you been reading cookbooks, and collecting recipes? (2) Are you now actually saving money for food at the end of the experiment so that you won't have to worry about expenses then? Out of 34 men, 19 answered the first question affirmatively, 15 negatively. But some of the "no" answers could

not be interpreted as showing a lack of interest in culinary matters; the subjects simply found other ways to express the interest. Thus one subject, a native of California, who did not read cookbooks industriously compared the prices of food, especially of fruit and vegetables, as advertised in California and Minnesota newspapers.

The second question was answered in the affirmative by 11 men, in the negative by 23. One subject answered "no" to this question though his answer to the first question was "yes," but he added, "I have money." In another instance of a negative answer the subject commented, "I wish I could save some money for that purpose and had thought of it."

As a result of the increased concern about food some men even went so far as to replan their lives. For example, one man became impressed by the importance of efficient methods of food raising and decided to go into agriculture as a vocation. A few planned to become cooks. Halfway through starvation 13 out of 34 men mentioned cooking among their plans following completion of the experiment. The men who took part in Greely's expedition of 1881 and suffered from prolonged semi-starvation in the Arctic had similar ideas. In the diary entry for November 24, 1883, Brainard (1929, p. 148) wrote: "Fredericks is going to run a saloon in Minneapolis. Long wants to open a restaurant at Ann Arbor. Jewell thinks he would like to run the grocery in Ralston's colony [to be established in Kansas]."

The idea of working as a cook was a real temptation to many subjects in the Minnesota Experiment. It may be instructive to include a paragraph from one of the reports on post-starvation plans. This particular subject listed as his first project choices relief work in Central Europe or in the Virgin Islands or a return to a west coast camp operated under the Civilian Public Service program. However, working as a cook was not foreign to his daydreams even though he commented, with his characteristic sarcastic humor, that he would not cook unless he could choose the spot and name the assistant cooks. He continued: "That cooking business is just gum beating — but, a big thick slice of Missoula ham with three or four eggs (whites firm but not hard, and yolks runny but cooked, please), and stack of medium brown toast with unlimited butter, to say nothing of a pitcher of milk and some coffee is not a possibility to turn aside lightly, and cooks get those things!" The language certainly reveals the sensuous pleasure of our semi-starved would-be gourmet.

In a few men there appeared, particularly toward the end of the experiment, a reaction against the "tyranny of food"; they became annoyed by discussions of food and related subjects. One man expressed disgust at this "animal attitude"; another referred to such engrossment as "nutritional masturbation."

Various techniques were developed to approximate or substitute for the satisfactions normally derived from eating. Prominent among these were gum chewing and smoking. The men often reported that they got a vivid vicarious pleasure from watching other persons eat or from just smelling food. Large quantities of water were consumed with and between meals, and the subjects increased the bulk of their food by "souping." For example, a man would drink the fluid from his soup, then fill the bowl with hot water, salt it heavily, drink the fluid off

again, and repeat this process before eating the solid part of the soup. Satisfaction was also obtained from consumption of coffee and tea (without cream or sugar but with a limited amount of saccharin), both of which were used in large quantities, presumably for their pharmacological as well as their filling and warming effects. It was generally reported that coffee and tea provided a "lift." Because some of the men increased their consumption to 15 or more cups daily, it became necessary to limit all subjects to a maximum of 9 cups per day. A few occasionally violated the spirit of this restriction by brewing the strongest possible tea or coffee and then diluting it further with hot water. About half a dozen subjects who had never drunk coffee or tea before the experiment became habitual users of both. For experimental purposes, the subjects were occasionally placed for 3-day periods on a severely limited fluid intake; when thus deprived of their beverages, a few complained of headaches and increased lassitude.

During the early part of the experiment the use of chewing gum was not limited; however, chewing rapidly became excessive. Heavy gum chewers would take 2 or 3 sticks at a time, chew them until the sweet taste was gone, discard them, and then replace them with fresh sticks in chain fashion. One of the men chewed up to 40 packages of gum per day and developed a sore mouth from such continuous exercise. Thereafter, the use of gum was restricted to 2 packages a day. Several men who had not previously used tobacco acquired the habit of smoking during the experiment because it afforded some degree of relief from hunger.

During the 12 weeks of rehabilitation there was relatively little change in the subjects' eating habits and attitudes toward food. The men continued to want more than they received. Even when those on the highest caloric intake were physically full they wanted more — their appetites were insatiable. The men continued to be concerned with food and their rations above all else. Food substitution habits persisted with only minor alterations. The heavy use of coffee and tea, the souping of food, and the generally high fluid intake continued to be characteristic for most of the rehabilitation period. The desire for hot food, the "formalities" of serving and eating, and the creation of mixtures and concoctions continued. About half a dozen of the men deteriorated more in their eating habits and table manners during the first 6 weeks of the rehabilitation period than during semi-starvation. There was little flagging of interest in food and culinary matters except for a gradual decline in filing recipes and reading cook-books.

Emotions and Attitudes

The cumulative stresses of semi-starvation resulted in emotional instability. The men experienced transitory and sometimes protracted periods of depression. They became discouraged because of their relative ineffectiveness in daily living. Inability to sustain mental or physical effort contributed much to this feeling of inefficiency. The persistent clamor of hunger distracted the subjects when they attempted to continue their cultural interests, manual activities, and studies. The discrepancy between what they wanted to do and what they were able to do was a source of serious frustrations. During the advanced stages of semi-starvation there was a marked lowering of the threshold to depressive reactions. Although

clinically the depression reached definitely pathological proportions on rare occasions and in only a few individuals, the men were almost always more serious and obviously less happy than during the control period.

Some spells of elation occurred. The men's spirits were markedly boosted by such things as good weather, anticipation of an outing, a stimulating "bull session," and other variations in daily routine capable of arousing enough interest or enthusiasm to take them "out" of themselves. Feeling "high" was sometimes attributed by the men to a "quickening" effect of starvation or to success in adjusting to the semi-starvation diet. These feelings of well-being and exhilaration lasted from a few hours to several days but were inevitably followed by "low" periods.

Both subjects and observers frequently remarked that the group, lively and responsive during the control period, became apathetic during starvation. In part, the apathy grew out of repeated failure, with accompanying frustrations, to carry on "normally" during the stress. On the other hand, things that would arouse their interest tended more and more to narrow down to the complex of "guinea pig" life: loss of body weight, hunger, and food. In discussing these, the men would often become animated.

The even-temperedness, patience, and tolerance evidenced during the control period gave way under stress. Irritability increased to the point that it became an individual and group problem. Although the men were well aware of their hyperirritability, they were not altogether able to control their emotionally charged responses; outbursts of temper and periods of sulking and pique were not uncommon. A few had strong urges toward violence, but these were controlled. The men who showed a large degree of personal and social deterioration became objects of aggression for the rest of the group. One of these subjects, who dramatized himself and his semi-starvation role, became in many respects the scapegoat of the group and a ready reference point for favorable self-comparison. The subjects considered themselves in comparison with their normal condition as lacking in self-discipline and self-control, indecisive, restless, sensitive to noise, unable to concentrate, and markedly nervous. It is likely that the widespread use of coffee, tea, and chewing gum represented attempts to allay nervous tension besides serving as substitutes for food. Similarly, the occurrence of nail-biting (not present during standardization) and the acquisition of or increase in smoking habits were related to increased nervousness.

Personal appearance and grooming began to deteriorate as the stress progressed. The men often neglected to shave, brush their teeth, and comb their hair. Even those who had been meticulous in their grooming now dressed carelessly and presented a slovenly appearance. Bathing, however, was not neglected; it was a source of pleasure since it was a means of getting warm as well as a form of hydrotherapy which was reported to have relieved aches, pains, and fatigue.

Social initiative especially, and sociability in general, underwent a remarkable change. The men became reluctant to plan activities, to make decisions, and to participate in group activities. Their earlier active interest in having a voice in the making of policies and rules for the conduct of the non-scientific as-

pects of the experiment dwindled. They spent more and more time alone. It became "too much trouble" or "too tiring" to have to contend with other people. With the decline in the interests which had previously been held in common with others and with the growth of feelings of social inadequacy, the men became self-centered. Because of this egocentricity and the associated heightened irritability, of which the subjects were well aware, it required at times a real effort for them to maintain socially acceptable behavior. Attempts to keep the interpersonal relationships tolerable, if not gracious, often produced an uncomfortable and emotionally charged atmosphere in which politeness was artificial and social interaction stilted. The humor and high spirits which had been an outstanding quality of the group during standardization gradually disappeared. The tone of the group became sober and serious. What humor remained at the end of semi-starvation was mainly of the sarcastic variety.

As mentioned earlier, the men devoted much time and energy to collecting recipes, studying cookbooks, contemplating menus, brewing their beverages, and fixing their "sandwiches." The acquisition of coffeepots, hot plates, kitchen utensils, and the like on shopping sprees appeared as a reasonable extension of these interests. Much less reasonable was the buying of old books, unnecessary secondhand clothes, knickknacks, and other "junk." Often after making such purchases, which could be afforded only with sacrifice, the men would be puzzled as to why they had bought such more or less useless articles. This acquisitive behavior can be interpreted as compensation for the deprivation of food. Several subjects insisted that they had grown unusually anxious to save money for a "rainy day" and attributed this to the insecurity they felt in the experimental situation.

In the later part of the semi-starvation period housekeeping chores were neglected and nonexperimental laboratory duties were carried out less and less effectively. The educational program designed to prepare the men for foreign relief work, and followed at the start with enthusiasm, in time quietly collapsed.

For 6 months the men had looked forward to the last day of semi-starvation as the day that would mark the end of their ordeal. They had anticipated that rehabilitation would bring about an early alleviation of their symptoms and distress. This belief was a sustaining motivation during the semi-starvation period. But the expected "new lease on life" did not materialize. The weight gains in the first weeks were small or nonexistent (a few men even lost weight owing to the loss of edema fluid). Weakness, tiredness, pains, and a myriad of other discomforts continued. Hunger and appetite were not appeased. As one subject expressed it, "Now I go away from meals hungry three times a day instead of two." Some men reported that their hunger pangs seemed more intense than ever before.

In the first 6 weeks of the rehabilitation period the spirits of the men, especially of those in the two bottom caloric groups (Z and L), were low. They were disappointed at not being rehabilitated at the anticipated rate. Some of them actually became more depressed and irritable than in semi-starvation. Many grew argumentative and negativistic. Others professed grave doubts as to the value of the entire project or questioned the motives and competence of the ex-

perimenters. Several subjects confessed that their humanitarian concern for the welfare of mankind had become elusive and difficult to maintain. Impatience, tenseness, and a feeling of being "let down" pervaded the group. After the first few encouraging days at the start of the rehabilitation regimen, there was a pronounced slump in morale.

However, the discontent and aggressiveness, as distinguished from the apathy and acquiescence of semi-starvation, was indirect evidence of an increase in energy. It preceded the gradual reappearance of physical, social and cultural interests which had gone "underground" for the duration of semi-starvation. No longer were the men willing to accept without question the formation of policy by those responsible for conducting the experiment. They insisted that the rules and regulations make good sense to them. The pronounced tendency toward introversion, so characteristic of the semi-starvation behavior, decreased early in the rehabilitation period. The men became more interested in their environment and exhibited annoyance at some of the restrictions that the experimental regimen continued to impose. They became increasingly impatient with the "buddy system," which was maintained until the sixth week of rehabilitation, when it was finally removed in the face of imminent wholesale violation. General unrest was reflected in the failure to re-establish nonexperimental laboratory and work assignments.

It was difficult for the men to abandon or modify attitudes and habits which they had acquired during semi-starvation. In returning to pre-experiment interests and activities, they were often frustrated by the lack of supporting strength and endurance. The circumstances of normal living appealed to the subjects long before they were able to cope with them successfully. This added to their restlessness and complicated the morale problem. It was only in the later part of rehabilitation that the cumulative effects of increased food intake and the approach of the end of the experiment brought about a noticeable improvement in morale. A gradual loss of lethargy and an increase in ambition, in the will to do things, were among the obvious indications of rehabilitation. The subjects no longer felt like "old men" and their increased energy was reflected in "useless" motions, such as more lively gestures when speaking.

Humor, enthusiasm, and sociability progressively reappeared; irritability and nervousness diminished. The sense of group identity, which became quite strong during starvation, was dissipated as the men once again looked forward to developing their own plans for the future and took up interests not immediately related to their participation in the experimental program. The distinction between the "in-group" and the "out-group" (i.e., the starved and the well-fed) was disappearing.

Dreams

Field reports contain references to the increased frequency of food dreams under conditions of food deprivation. Sorokin (1942, p. 29) states somewhat dogmatically that the dreams of hungry persons are occupied primarily with food. The validity of all such statements is rather doubtful since detailed information is never given on the total number of dreams and their topics. The con-

cern with food during the waking hours would act as a selective factor, and there is a better chance that food dreams would be remembered or at least would be more talked about than other dreams. This is in line with Petrajitsky's statement, quoted by Sorokin (1942), that in starvation the attention tends to be strongly attracted toward food phenomena.

In the group studied under experimental conditions by Miles (in Benedict *et al.*, 1919) 4 out of 12 men in Squad A, maintained on a reduced intake for 4 months, reported in post-experimental interviews that they had had food dreams; only 2 men of Squad B, maintained on the restricted dietary regimen for 3 weeks, recalled having dreams of food. Prominent in the dreams was some situation in which the men were about to break the dietary restrictions. Methodologically, Miles' approach was not completely satisfactory; recalling dreams over a period of weeks or months is an unreliable procedure.

TABLE 363

FREQUENCY OF DREAMS DURING SEMI-STARVATION AND REHABILITATION IN THE MINNESOTA EXPERIMENT. S8, S11, S12, and S20 = 8, 11, 12, and 20 weeks of semi-starvation; R7 = 7 weeks of rehabilitation.

Date	Week	Number of Subjects	No Dreams	Food Dreams	All Other Dreams
4/9/45	S8	36	26	0	10
5/2/45	S12	35	19	3	13
7/5/45	S20	34	20	1	13
9/17/45	R7	32	22	1	9

Because of the obvious relevance of the problem, an attempt was made in the Minnesota Experiment to avoid the bias present in the earlier observations. At intervals during the experiment the men were asked in the course of a morning testing session to describe the dreams they had had during the night. These surveys were not announced in advance and approximated a truly random sampling of the dreams. The results, tabulated in Table 363, are in striking contrast with such statements as that of Sorokin. Food dreams were rare and there was no marked tendency for them to increase as starvation progressed in the Minnesota Experiment; in rehabilitation the pattern did not change. On reading the diaries of at least some of the subjects one might easily obtain just the opposite impression, but the diary records obviously present a selected rather than a random sampling of the content of the dreams.

Sex

Sexual feelings and expression declined in the Minnesota Experiment until by the end of the semi-starvation period they were virtually extinguished in all but a few subjects. The diminution of the strength of the sex drive was so dramatic that the subjects were struck by the change and used colorful language to describe it. As one of them put it, "I have no more sexual feeling than a sick oyster." The number of dates dropped drastically. Those who continued to date found their relationships strained. Some of the men were surprised to find that

this was true even where their female contacts had appeared to be based on intellectual interests. Such situations probably stemmed from a decline in sociability as well as from the loss of sex impulses. Masturbation and nocturnal emissions ceased or were greatly reduced in frequency. Sex fantasies and sex dreams were reported to have decreased in number, and when present they were much attenuated. In the rehabilitation period, sexual impulses, needs, and interests were very slow in regaining their pre-experiment intensity; they were still low at the end of the twelfth week of rehabilitation.

The loss of the sex drive in semi-starvation is not an unmixed "evil." Biologically it may be considered one of the adaptive mechanisms protecting the individual organism from nonessential energy expenditure. Psychologically many of the men welcomed the freedom from the sex tensions and frustration normally present in young adult men. A number of men who had religious scruples against the practice of masturbation but were unable to control sex tensions in other ways were relieved at the cessation of the impulse to engage in this activity.

It can be postulated with confidence that in part the changes in the sex drive had a physical basis in the decreased hormonal function of the testes. It is known from animal anatomical studies that the testes show a pronounced loss of weight. Parsons (1939, p. 96), basing his figures on the older experiments of Voit, indicated that in starvation the weight loss of the testes, expressed as a percentage of the initial weight, was about 40 per cent, roughly midway between that of the brain and the adipose tissue (stored fat), for which the relative weight decrements were given as 3 per cent and 97 per cent, respectively. There are also data on man indicating that the testes decrease markedly in undernutrition; in 27 cadavers of tuberculous patients whose average body weight was 43 per cent below the estimated "standard," the calculated loss of weight of the testes was 49 per cent (Krieger, 1921, p. 117). In view of these striking morphological changes, one would expect also profound functional alterations. In the Minnesota Experiment no attempts were made to determine the degree of atrophy but investigations of the sperm showed marked changes (see Chapter 35). There was also a decrement in the excretion of 17-ketosteroids.

The effect of the lowered nutritional level on the sex drive was observed in the study by Benedict *et al.* (1919) and was reported in detail by Miles in a separate publication (Miles, 1919). The information was obtained in interviews carried out after the men were released from dietary restrictions. The material was presented in the form of excerpts from the interviews. General sex interest was reported by nearly all the subjects to have decreased. There seemed to be a definite decrease in the frequency of nocturnal emissions and erections. Information on other aspects of sex expression was fragmentary.

In this context a reference may be made to Biskind's study (1947) on the relationship of nutritional deficiency to impaired libido and potency. In a group of 143 patients, 29 to 61 years of age, with vitamin B complex deficiency, two thirds were aware of sexual inadequacy. Improvement by diet alone was reported by 62 out of 76 patients. It is only fair to remark, however, that the possible effects of psychotherapy and suggestion were not evaluated.

Dietary Factors in Rehabilitation

In the Minnesota Experiment the number of complaints and undesirable symptoms per subject increased, on the average, from 6.6 during control to 20.5 at the end of semi-starvation. This represents an increase of 13.9 points above the control value. By the middle of rehabilitation (R6) the recovery in regard to complaints amounted to 37 per cent of the semi-starvation deterioration; by the end of the twelfth week of rehabilitation the recovery had increased, on the average, to 66 per cent. There was some tendency for the two bottom and the two top caloric groups to differ in the amount of recovery, but the lower (Z vs. L) and upper (G vs. T) groups were not differentiated at all (see Table 364).

TABLE 364

COMPLAINTS IN THE MINNESOTA EXPERIMENT; increase in the number of complaints above the control values. S12 and S24 = 12 and 24 weeks of semi-starvation; R6 and R12 = 6 and 12 weeks of rehabilitation. N = 32.

Caloric Group	S12	S24	R6	R12	S24 - R12	(S24 - R12) S24	× 100
All groups combined ..	8.5	13.9	8.7	4.7	-9.2		66
Z (basal)	6.8	13.0	8.7	5.8	-7.2		55
L (+400 Cal.)	6.9	10.6	10.7	5.3	-5.3		50
G (+800 Cal.)	9.3	15.9	7.6	3.6	-12.3		77
T (+1200 Cal.)	9.8	16.0	7.8	3.9	-12.1		76

When the men were asked to estimate the percentage of their over-all rehabilitation there was a tendency for the caloric groups to be separated both at the sixth and, even in a more pronounced way, at the twelfth week of rehabilitation (see Table 365). There were only negligible differences between the men who did and those who did not receive protein or vitamin supplements.

The third criterion of deterioration in semi-starvation and of recovery in rehabilitation was provided by the intersubject ratings. The score for each man is the average of the several deterioration ratings given him, independently of each other, by a group of his fellow subjects. For the group as a whole (see Table

TABLE 365

REHABILITATION IN THE MINNESOTA EXPERIMENT; self-ratings of over-all improvement from semi-starvation deterioration. The values indicate the estimated percentage of rehabilitation; full recovery = 100 per cent. R6 and R12 = 6 and 12 weeks of rehabilitation.

Group	R6	R12	Group	R6	R12
Caloric			Protein		
All groups combined	41	68	U (basal)	40	68
Z (basal)	33	59	Y (extra)	42	68
L (+400 Cal.) ...	36	64	Vitamin		
G (+800 Cal.) ...	46	68	P (basal)	44	68
T (+1200 Cal.) ..	49	81	H (extra)	38	68

TABLE 366

INTERSUBJECT RATINGS IN THE MINNESOTA EXPERIMENT. The over-all deterioration was rated on a scale from 0 (no deterioration) to 5 (extreme deterioration). Each man was rated by the same group of subjects throughout, and his individual score at each period was the average of the several independent ratings. The values given here are the averages of the individual scores in the indicated groups. S12 and S24 = 12 and 24 weeks of semi-starvation; R6 and R12 = 6 and 12 weeks of rehabilitation.

Group	S12	S24	R6	R12	(S24 — R6)	(S24 — R12)	(S24 — R6)	(S24 — R12)
							S24	S24
Caloric								
All groups combined	1.6	2.3	1.4	0.7	—0.9	—1.6	.39	.70
Z (basal)	1.5	2.2	1.5	0.9	—0.7	—1.3	.32	.59
L (+400 Cal.)	1.7	2.4	1.7	0.8	—0.7	—1.6	.29	.67
G (+800 Cal.)	1.5	2.5	1.2	0.6	—1.3	—1.9	.52	.76
T (+1200 Cal.)	1.6	2.2	1.0	0.4	—1.2	—1.8	.55	.82
Protein								
U (basal)		2.2	1.4	0.7	—0.8	—1.5	.36	.68
Y (extra)		2.5	1.4	0.7	—1.1	—1.8	.44	.72
Vitamin								
P (basal)		2.3	1.3	0.7	—1.0	—1.6	.43	.70
H (extra)		2.4	1.5	0.7	—0.9	—1.7	.38	.70

TABLE 367

SIGNIFICANCE OF THE DIFFERENCES IN INTERSUBJECT RATINGS OF OVER-ALL DETERIORATION IN DIFFERENT CALORIC GROUPS AT R12. V_{rep} (replicate variance) = 0.27. (Minnesota Experiment.)

	V_{bGr}	F
Z vs. L	0.25	
Z vs. G	1.76	6.41[*]
Z vs. T	1.32	4.81[*]
L vs. G	0.68	
L vs. T	0.42	
G vs. T	0.03	
(Z + L) vs. (G + T)	1.95	7.10[*]

366) there was an average recovery of 39 per cent at R6 and of 70 per cent at R12; this compares fairly well with the results of the complaint inventory and of the subjective estimates of over-all recovery. The trend of recovery closely paralleled the levels of caloric refeeding. A statistically significant differentiation was obtained by comparing the means of the lower and upper two caloric groups (see Table 367); also, recovery in the Z group was significantly less than in groups G and T. The protein and vitamin groups were not differentiated.

Behavior after Release from Dietary Restrictions

At the end of the twelfth week of controlled rehabilitation, 20 of the 32 subjects were released altogether from dietary and experimental restrictions. The 12 men selected to remain at the Laboratory for further study were relieved of all dietary restraints over weekends. After repeated warnings and instructions

about overeating and indulgence in long-forbidden foods, the restrictions were removed at noon on Saturday, October 20, 1945, and a special farewell dinner was served at 5:00 P.M. The urgent desire for dietary freedom expressed by the men was extreme; postponement for another week could have produced severe emotional crises and possibly open rebellion. All the men were counting the hours until they could have more food, and this intense concern was scarcely less in the men who had been on the most liberal (T) rehabilitation diet, which had averaged 3392 Cal. daily for 12 weeks and 4014 Cal. for the 2 weeks immediately preceding release.

The farewell dinner was really a banquet, in both quantity and quality, but moderation was advised in most serious terms. The majority of the men found they were surfeited with considerably less food than they had expected, and several men had to leave the table because they developed stomach pains and distention. The end of the meal found most of the men gazing in unbelief at the food — their food — which they could not eat.

The week of R13 was largely devoted to eating and sleeping. Many men ate more or less continuously. Some reported eating as many as three consecutive lunches. In many cases the men were not content to eat "normal" menus but persevered in their habits of making fantastic concoctions and combinations. The free choice of ingredients, moreover, stimulated "creative" and "experimental" playing with food. Generally the men ate more food than they were prepared to cope with. This gluttony resulted in a high incidence of headaches, gastrointestinal distress, and unusual sleepiness. Several men had spells of nausea and vomiting. One man required aspiration and hospitalization for several days.

The licking of plates and neglect of table manners persisted. Attempts to avoid wasting even a particle of food continued in the face of unlimited supplies. An irrational fear that food would not be available or that the opportunity to eat would somehow be taken away from them was present in some of the men. This may have motivated their eating all they could hold at one time.

The voluntary caloric intakes of the 12 men remaining at the Laboratory for further study were carefully measured during this week. These averaged from 4360 to 5770 Cal. per day for the different individuals, and the grand average for the 12 men was 5218 Cal. daily for the week. Murray (1947) studied 930 men after 2 to 3 weeks of preliminary hospital treatment following release from German concentration camps. The estimated weight loss amounted to 23 per cent of the original body weight. After the first few days of restricted feeding the average daily food intake rose to the prodigious level of almost 8000 Cal. (297 gm. protein, 955 gm. carbohydrate, 281 gm. fat).

Many Minnesota subjects commented that they were still "hungry," though incapable of ingesting more food, at the end of the very large meals that were the rule. All the men ate snacks between meals and in the evening. There were no sharply defined predilections for particular foods, though milk in one form or another was taken in remarkable quantities. The consumption of fruits and nuts was limited somewhat, in accordance with the urgent advice of the Staff.

After R12 the diaries and systematic interviews in the Minnesota Experiment were discontinued and were replaced by summary reports written by the sub-

jects for the thirteenth, fifteenth, and twentieth weeks of rehabilitation; at the follow-up examination in March (at R33) a short personal interview was held. In the reports the subjects were asked to comment on their complaints, eating habits, cravings, the quantity of food eaten, the importance of food, the ability to do physical work, and their general adjustment.

The material for each man was transcribed in an abbreviated form on separate sheets, using one page for one category at one time interval. In each category a few broad classes were defined; for example, in the category of complaints there were "no complaint," "gastrointestinal disturbances," "sleepiness," "headache," "edema," and "miscellaneous complaints." The frequency numbers (i.e., the number of men falling into each class) were determined. Case illustrations will be used to make the presentation of these data more concrete and to indicate the limits which have to be placed on any generalization of the symptoms. In this way we attempt to combine the quantitative characterization of the group with a more discursive, but also a more descriptive, case material.

From the point of view of mechanics, the use of the written reports, with the material separated into well-defined categories, was more efficient than the diaries and interviews, which were very difficult to summarize. Because the process of recovery provides a good frame of reference for the changes that took place during semi-starvation, the material will be presented in some detail.

Complaints after R12

During the first week following the release from dietary limitations (R13, October 21-27), gastrointestinal disturbances such as stomach pains, "gas," flatus, belching, heartburn, and discomfort from overeating were experienced singly or in combination by 23 out of 28 men for whom complete records were available. There was some constipation (3 men), and one man reported diarrhea. In another subject, a large number of bulky bowel movements left the rectum and anus quite sore. Sleepiness was a frequent complaint (10 men), and headache had a similar incidence. Tiredness was reported by about two thirds of the subjects. Three men complained of excessive thirst and an inadequate amount of saliva. There was noticeable edema in No. 1 (face) and No. 108 (ankles); in No. 29 the edema in the knees was greater than at any other time. Four men commented that they did not have to get up any more at night to urinate. There was some muscle soreness (3 men). No. 29 still had a "crawling" feeling on the upper leg.

By the week of November 4-10 (R15) the incidence of gastrointestinal complaints remained high (15 out of 30 men reporting), with little change in the type of complaints. Unusual sleepiness was reported by 12 men. Five men had clinical edema: in No. 1 the edema fluctuated, came and went; in No. 108 it was still pronounced, and No. 120 had more evident edema than in semi-starvation; the edema was marked in No. 29, and No. 122's feet were swollen. In No. 29 the paresthesias in the upper leg were still present. No. 104 was more sensitive to heat than he had been in semi-starvation; he could not drink his tea as hot as before.

In the later part of December (R20) 13 out of 28 men reporting had no com-

plaints. Gastrointestinal difficulties, especially intestinal distention, belching, and flatus, were still experienced by 8 men. No. 120 had an upset stomach and diarrhea several times, which he ascribed to anxiety and speed of eating, as well as to some overeating. No. 19 still had to get up at night to urinate — a semi-starvation symptom that had long since disappeared in the majority of the men. In 3 men (Nos. 111, 9, and 108) obesity was the main complaint. No. 119 had edema in the legs and ankles. In No. 29 the swelling in the ankles and knees had decreased somewhat, but his knees were still weak and to maintain a squatting position was quite a strain; a sign of rehabilitation to this man was the return of sensitivity to an area of the leg that had become "numb" in semi-starvation. No. 101 was still easily irritated by noise, and the proximity of other individuals also bothered him. No. 130 was getting back close to normal but still was "on the nervous side."

In March (R33) most of the 16 men for whom we have a good record had no complaint except for shortness of breath (Nos. 122, 130, 129, and 123). No. 4 retained some physical lethargy. In No. 130 there was still some edema. Many commented that their hair was no longer falling out as in semi-starvation (and early rehabilitation) and that it was beginning to grow more abundantly.

Eating Habits after R12

During the latter part of October (R13) only 3 of 26 subjects who mentioned anything about eating habits characterized them as "normal." A few thought their manners were almost conventional again but qualified their statement, especially with reference to the speed of eating. Some men continued to eat slowly as they had during semi-starvation; No. 111 and No. 27 took a longer time to eat than before the experiment; No. 122 felt he was "gulping" his food when he kept pace with other people; No. 19 ate very slowly even though he disclaimed a desire to play with food. Many noticed an improvement in table manners; No. 20 began to observe the niceties of eating — no eating with fingers, and one dish at a time; No. 104 did not lick his plate any more but still wiped it with bread; No. 26 also cleaned his plate thoroughly, and No. 4, in addition, picked up crumbs. No. 109 considered his table manners still somewhat rough. No. 101 remarked that his manners were forced back closer to normal by the desire to "act decent" in the Student Union. No. 12 tended to use his spoon instead of a fork and was occasionally tempted to lick butter from his knife. No. 29 strove consciously to discipline his manners, but No. 119 had little concern for the niceties of eating and still mixed his food as in semi-starvation. No. 5 picked up crumbs and on one occasion licked the cream off the cap of a milk bottle. No. 30 preferred to concentrate on eating, not on conversation; No. 23 preferred to eat alone for the same reason.

By the second week of November (R15) the number of normal and near-normal ratings of table manners increased to 19 of the 26 subjects reporting. While others reported good table manners, their speed of eating was still not normal. No. 108 ate faster than the rest of his family in order to eat enough within the conventional mealtime. No. 11 also ate rather fast; he "gobbled up" his food, and scraped his plate clean. No. 5 continued to scrape dishes with his

spoon and had a desire to lick plates. No. 20 still took a long time to eat. Subject No. 122 shared this starvation habit; he licked his knife when eating alone, and at the table he was irritated at not being able to get his plate clean with the ordinary utensils. When the food was liquid, subject No. 29 wiped the dish with bread. Extreme disregard of the conventional eating manners was rare. No. 119 had no inhibitions about gnawing meat from bones, making "fancy" sandwiches, or mixing food at the Student Union Cafeteria.

In the later part of December (R20) all men (24 reporting) rated their eating manners as normal or nearly normal. Some of the minor "misdemeanors" were taking larger bites (No. 1); eating everything on the plate, even the celery tops (No. 11); and mopping the plate with bread (No. 29). Subject No. 119, who in November freely disregarded eating conventions, now reported a decreased preoccupation with food and a more social behavior at meals. Four subjects noted that they still were eating more slowly than other people.

In March (R33) either there were no comments on eating habits or they were characterized as normal.

Cravings after R12

During the week of October 21-27 (R13), of 17 men who made a reference to food cravings, 7 had no specific cravings. The remaining 10 men had a predilection, in about equal proportion, for sweets (ice cream and pastries) and dairy products (milk, eggs, and cheese, also nuts). The opportunity to choose their own foods gave some men a feeling of release from the long-continued adherence to controlled diet. One man (No. 123), who continued to reside at the Laboratory and ate fixed meals on weekdays, did not have particular food cravings, yet he experienced an exhilarating sense of freedom on weekends when he was not bound in any way by the diet. On the other hand, for some men the selection of things to eat was a bother, an unwanted responsibility; No. 5 preferred that others decide about the food to be served.

During November 4-10 (R15) 18 men out of 26 reporting had no unsatisfied cravings. Sweets were the favorites of 8 men; No. 122 could not get enough ice cream to satisfy his appetite, and No. 11 ate as much ice cream as he could "lay his hands on" but commented that *all* food tasted good and that each meal was a real treat. Of the dairy products, milk was mentioned most frequently (5 men).

In December (at R20) 21 out of 25 men reported "No cravings." Of the other 4 men, No. 122 craved nuts, Nos. 27 and 11 craved milk and eggs, and No. 26 kept on eating two chocolate bars a day until February.

Quantity of Food Eaten after R12

Out of 17 men who did not stay in the Laboratory but who made reports on their food intake for R13, 15 apparently ate from 50 per cent to over 200 per cent more than during the eleventh and twelfth weeks of rehabilitation; only one reported that his intake was the same, and one man (No. 11) ate less. "Snacking" was prevalent: No. 102 ate immense meals (a daily estimate of 5000-6000 Cal.) and yet started "snacking" an hour after he finished a meal. No. 122 ate as much as he could hold during the three regular meals and ate snacks in the morning, afternoon, and evening. The men frequently found it difficult to stop

eating. No. 20 "stuffs himself until he is bursting at the seams, to the point of being nearly sick," and still he felt hungry; No. 120 reported that he had to discipline himself to keep from eating so much as to become ill; No. 1 ate until he was uncomfortably full; and subject No. 30 had so little control over the mechanics of "piling it in" that he simply had to stay away from food, because he could not find a point of satiation even when he was "full to the gills." Subject No. 104 also wanted to eat more even though his stomach was filled.

The men who continued to stay at the Laboratory exhibited a marked tendency to increase their intake over the weekends when they were completely free to choose their food. "I ate practically all weekend," reported subject No. 26; No. 123 had a tendency to overeat during the weekends; No. 129, a subject with a strong sense of self-discipline, experienced uncomfortable overeating on two weekends (October 20 and October 27).

During the week of November 4-10 (R15) appetites remained keen. Many men were still stuffing themselves at meals and eating snacks between meals and at bedtime. Others were gradually getting away from overeating; for example, No. 105 still ate a lot of food but he was not the "hog" he had been. Of 17 men reporting, 9 were still eating more than during the weeks of R11 and R12, 3 much more, 3 about the same, and only 2 somewhat less.

The same picture was presented by the 12 men who stayed at the Laboratory. Three men were well satisfied with the regular Laboratory fare. One (No. 23) found the diet larger than he would prefer. Eight men would have eaten more in the regular meals if more had been available; No. 26 would just as soon have eaten six meals instead of three. Most men ate extra sandwiches and milkshakes during the week and considerably more on weekends.

By December (R20) 10 out of the 15 men who reported had reduced their food intake to or below that of R11 and R12. Subject No. 105 even omitted breakfast so that he could lose weight and No. 5 was limiting his intake to stabilize his weight at 160 lbs. (pre-starvation weight, 178 lbs.). A few men continued to consume prodigious quantities of food: No. 108 would eat and eat until he could hardly swallow any more and then he felt like eating again half an hour later; No. 122's intake was still estimated at double that of R11 and R12, No. 9's at about $1\frac{1}{2}$ times that amount, and No. 22's at about $1\frac{1}{2}$; No. 8 was still eating more than normal and still gaining weight. In general, the men who remained at the Laboratory were rapidly approaching normal food intakes and were satisfied with progressively less food. Their interest in outside eating decreased, and the pattern of weekend gorging largely disappeared.

The majority of men (10 out of 14) interviewed in March (R33) reported eating normal amounts of food at meals, either without (5 men) or with (5 men) snacks between meals; few ate more than before the starvation experiment. Subject No. 9 ate about 25 per cent more than his pre-starvation amount; once he started to reduce but got so hungry he could not stand it.

Importance of Food after R12

During the thirteenth week of rehabilitation food was still a major concern and interest for 15 out of 24 men who mentioned this item in their report. One

man (No. 102) was still very much concerned with food; another (No. 108) stated that eating continued to be his most pleasurable activity. Subject No. 29 suffered from an anxiety that "the food might not last," and he kept on hand a small stock of bread, fruit, and sausage; only gradually was he becoming interested also in the people with whom he ate. For 9 out of 24 men food was decreasing in importance; it did not weigh on their minds as it used to. Subject No. 111 reported that his appetite was still keen but that food was losing its prime importance in life; No. 22 thought seldom of food; for No. 123 food was no longer an exclusive concern; according to No. 11 food did not monopolize his conversation to the extent it had before and, surprisingly, seeing food wasted no longer bothered him.

At the beginning of November (R15) 14 out of 24 men reporting still considered food and eating a much more important item in life than they had before the experiment; for No. 30 food was still of first importance — occasionally he felt a "slave" to food; No. 22 frequently ate more than necessary, partly for the pleasure of eating and partly to prevent waste. Fear that the leftovers might be thrown away was nearly obsessive in No. 102, who felt that he had to consume any uneaten food whether he was hungry or not; No. 104 still thought a lot about food and would rather eat than go to a concert or study; No. 8 helped his mother with the cooking; for No. 108 eating continued to be a favorite pastime, and he did some baking and cooking. In some men (e.g. No. 111) the preoccupation with food was narrowed down to the time before meals; for No. 20 food was still a central point of thought, but he put more emphasis on foreign relief and less on his own "belly"; for No. 11 food continued to be important and colored his thinking on political and economic matters.

The remaining 10 subjects reported normal or nearly normal attitudes toward food at R15: No. 27 had no thought of food until mealtime; No. 2 at times regretted the necessity of having to leave his work to go out for a meal; for No. 101 eating was becoming a routine matter, something that had to be done; for No. 9 meals were no longer a ceremonial ritual; for No. 29 food was no longer the focus of attention, and he did not feel anxiety when a meal was delayed; other interests, particularly those related to work and sex, overshadowed No. 127's interest in food.

In December (R20) food was still a vital concern and a center of interest for 11 out of 27 men. They continued to be more conscious of food than before the experiment (No. 26) and were aware of the quality as well as the quantity of foods eaten (Nos. 123, 4, and 19). A few were interested in the ways in which foods were prepared and frequently did cooking and baking themselves (Nos. 8 and 120). No. 122 was conscious of food ingredients and got a "kick" out of purchasing family groceries. In some men this concern was expressed as intolerance of food waste (Nos. 101 and 4). Subject No. 108 considered the facility and security of food provision in making his occupational plans and asked, "What and how much food can one get if working in that trade or service?" Subject No. 11 still got a thrill out of eating but was occasionally able to skip breakfast and stay in bed to get more sleep.

For the other 16 of the 27 men food decreased in importance to practically a

normal level by R20. Eating was merely one of the pleasant parts of life for No. 104, instead of being "all there is to live for." Subject No. 9 was no longer overconcerned with eating; he ate what was served when it was served and other interests occupied his mind to such an extent that he could even skip a meal if time taken for eating would interfere with other activities. No. 105 also was not bothered by skipping a meal, even though he had made a vow in semi-starvation never to miss any opportunity to eat. For No. 12 the regularity of meals was no longer essential. For No. 127 food no longer had a strong emotional appeal and was important only from the standpoint of sustaining life and providing energy.

By March (R33) the attitude toward food had returned in general very close to normal. Food was no longer *the* significant factor in life. The men were not much interested in food as such. Eating had become routine. Abhorrence of food waste was dropping. The desire of No. 122 to be around the kitchen was decreasing rapidly. The men still thought about food but in more impersonal terms, being aware of the importance for health of wholesome food in sufficient quantity.

Work Capacity after R12

At the end of October (R13) only 12 out of 29 men reporting felt fairly strong. No. 123 was able to haul and saw wood for 2 hours before becoming tired; physical work was attractive and pleasant. For No. 111 walking a distance of 2 miles and carrying about 30 lbs. of baggage had about the same effect as in "normal" times. No. 1's work capacity had improved so much that he could pitch stovewood and do chores without particular difficulty. No. 129 had ample energy and ran upstairs two steps at a time.

Different aspects of fitness were returning at different rates. No. 4 tired rapidly but his maximal strength seemed to be close to normal. No. 109 could ride a bicycle quite well but lifting heavy objects was still difficult. No. 104 held up pretty well in muscular work that did not require speed, such as carrying lockers and beds in moving the dormitory, but could not develop any speed when he tried to play football. No. 101 enjoyed moving laboratory equipment and furniture but was very sore and stiff afterward.

Of the 29 men reporting at R13, 17 felt unable to do hard physical work; when they attempted to work they felt weak, slow, and short-winded and tired easily. Lack of endurance was a prominent and frequent complaint. For No. 23 laundry work was fatiguing; in trying to chop wood he was not strong enough to swing the ax effectively, and he tired rapidly. Many found it difficult to carry their suitcases for any appreciable distance. No. 112 thought his muscles seemed soft; eating the tremendous quantities of food made him feel "heavy" and discouraged any thoughts of doing hard physical work. Subject No. 26 complained, "All this food is putting a stomach on me but that's all"; his gross body coordination was still poor as was demonstrated in his unsuccessful attempts to play football.

Two weeks later, at R15, the picture had not changed substantially; the ability to do hard work had increased only slightly. A large number of men still tired easily and their strength was below normal. The arm muscles appeared to be particularly slow in recovering. For example, No. 5, who worked as an orderly

in a hospital, was able to square dance but he could not lift patients as easily as he used to. No. 127's physical capacity was still below par; he could only chin himself 2 or 3 times as compared to his pre-starvation score of 16-18. No. 123 was able to push a car but was still unable to do even one push-up. In doing relatively light work, such as gardening, frequent rest periods were needed (Nos. 29 and 101). Subject No. 112 still lacked "ambition, stamina, and brute strength."

In December (R20) 15 out of 25 men reported a substantial improvement in their ability to do physical work. A number of men felt they were nearly back to normal (Nos. 1, 12, 112, 26, and 104). No. 111 was as able as ever to do hard physical work, including the chores and general farm work at home. No. 30 could lift cans and crates of milk bottles in a dairy just as easily as before the experiment. Other men felt strong but did not have "lasting power" (Nos. 122, 8, 29, 23, and 4). Ten men out of 25 still felt weak and lacked endurance. Subject No. 20 tired quickly when washing walls and moving furniture. For No. 19 it was still difficult to get out and do a full day's work without feeling quite tired; he commented that his arms were particular fatigable. Shoveling snow for an hour made No. 11 very tired and stiff. No. 120 still felt "soft" and could not do much work; except for attending church he was content to sit at home and visit with the family.

In March (at R33) the capacity for hard work was generally considered to be about back to normal, even though some men (Nos. 29, 4, 130, 123, and 19) did not do much physical work and did not exercise. Men who had done physical work had regained their full capacity. No. 122 was easily able to keep up with the men on his work crew in the camp, although it had taken him about 3 weeks (in February) to come back to normal. No. 1 had rapidly got used to the heavy work of putting out apple trees. No. 30 was doing hard physical work in a dairy without difficulty, and he played basketball and volleyball with relish. Most of the work in the woods caused No. 109 no difficulty; only in lifting heavy logs was he not back to normal. No. 9's strength was almost normal and his endurance was good, but being overweight, he tended to be less active than before the experiment. Carrying heavy loads still seemed somewhat harder than it used to be for No. 129.

Sex Drive after R12

Sex drive was not specifically mentioned in the follow-up questionnaires, but some men volunteered information about it. In October (at R13) No. 129's sex interest was quite low; on the other hand, No. 104 had his first nocturnal emission on October 22 and No. 29's sexual feelings were strengthened.

In November (at R15) No. 104 reported having two nocturnal emissions a week; once he had masturbated, but social contacts with girls were as yet not appealing. No. 101 was becoming interested in women. The sex drive had returned to near normal in Nos. 123, 122, 1, and 127. In No. 11 sex interests had gone way up.

In December (at R20) No. 5 reported a high sex drive which had become the dominant interest in his life. In No. 130 increased masturbation may have represented, according to his own comment, a sign of restlessness rather than of rehabilitation.

In March (at R33) 8 out of 12 men reporting considered their sex drive normal; 3 subjects rated their sex drive as strong or very strong; only in one case (No. 130) was sex interest still less intensive than before the experiment.

Effects of the Starvation Experiment on General Attitudes

In October (at R13) 14 men made comments on this topic. They reported an increased awareness of the role food plays in maintaining health (Nos. 123, 9, and 108) and an abhorrence of the waste of food (Nos. 123 and 22). Subject No. 5 became concerned with developing a sense of moral responsibility in people to provide adequate standards of living for everyone. No. 20 felt that the experience gained in the experiment had not modified his outlook in any fundamental way but that it had broadened his social thinking. In some men the long, strict adherence to the laboratory regimen produced a strong revolt; No. 30 wanted to be free, not to be a slave to food or any routine way or pattern of life. No. 123, who remained at the Laboratory, enjoyed an exhilarating sense of freedom on weekends when he was free to choose his own food. Some men felt an anxiety related to food; for example, No. 29 became irritable when the meal schedule could not be maintained. Others had a general sense of greater freedom; No. 11 was less worried about how to make a living after he saw that he was able to survive on so little food, and No. 19 had a more leisurely attitude toward life.

The November (R15) reports contained little that was new. Concern for the starving and the undernourished continued. There was an increased interest in larger social issues and a greater tolerance for other people's reactions and attitudes. No. 129 had more self-confidence and more feeling of security than before the experiment. No. 29 was more courageous, less inhibited, and more tolerant of others, especially of children and older people. No. 102 "took things more easy" and disliked the pressure of time. But the "rebellious" tendency was also present. No. 5 quit his job as a hospital attendant after a quarrel with the supervisor because he was "not willing to submit blindly to any authority." To No. 119, who remained in Minneapolis as a subject, the Laboratory still symbolized frustration and he would have preferred to leave if he could have.

In December (at R20) the sensitivity for starving people was still strong in the majority of the subjects, but some men had to remind themselves consciously to think of people who do not have enough to eat. No. 122 still resented food wastage but with decreasing vigor.

A Comment by One Subject

It may be useful to conclude this chapter by presenting verbatim a description of the experiment as seen by one of the subjects, a man of above average intelligence who had good insight into his condition and a gift for verbal expression. The report was written 8 months after his release from controlled diet:

"Will you Starve That They Be Better Fed? This was a dramatic question on the cover of a pamphlet issued by Civilian Public Service men at the Laboratory of Physiological Hygiene, University of Minnesota. It was a call for volunteers to be human guinea pigs in an experiment on semi-starvation and rehabilitation in man. Scientists wanted to know not only the consequences of semi-starvation

but the efficiency of calories, proteins, and vitamins in rebuilding starved bodies. The goal was a scientific foundation on which to base better relief methods in refeeding starved Europeans and other people of the world.

"I was working on a senile incontinent ward in a mental hospital in Concord, New Hampshire, when I first came in contact with "Will you Starve." For a long time I had been wondering how I could be of greater help in relieving misery in a war-torn world. This opportunity for service was one which would not require a college education, or special abilities. I had a sound body to offer — I would be starved — but only for six months and then I would be rebuilt.

"As one of the thirty-six 'lucky' ones chosen from several hundred volunteers, I arrived in Minneapolis on a rainy day in November [1944]. During the next three months I ate a normal diet and took numerous physical and mental tests. The test results obtained during this normal period were to be compared with those obtained during starvation. I enjoyed life in the new university atmosphere. Then came February twelfth, the starting date of semi-starvation. Only two meals a day from now on and only three daily menus to be rotated day after day for six months.

"For a few weeks the new life was fun. I was losing weight, of course, but I still had a lot of energy. Then came the day when I lost my 'will to activity.' I no longer cared to do anything that required energy, and days began to drag — each day getting longer and longer and there seemed no end of starvation in sight. Six months were an eternity.

"But they went by: slowly, slowly. I would compare my reflection in the mirror with that of my picture in pre-starvation days. My hair was thinner, eyes looked hollow, cheeks were only thin coverings for the bones of my face. When I tried to smile it was a grimace and I didn't feel like smiling and never laughed. My muscles were almost gone, my bones protruded (even a few minutes of sitting on a hard chair were uncomfortable) and my skin was dry and lusterless. A quarter of my original body weight had been consumed for energy during this period of deficiency. I felt as old as the aged men on my hospital ward at Concord.

"How does it feel to starve? It is something like this:

"I'm hungry. I'm always hungry — not like the hunger that comes when you miss lunch but a continual cry from the body for food. At times I can almost forget about it but there is nothing that can hold my interest for long. I wait for mealtime. When it comes I eat slowly and make the food last as long as possible. The menu never gets monotonous even if it is the same each day or is of poor quality. It is food and all food tastes good. Even dirty crusts of bread in the street look appetizing and I envy the fat pigeons picking at them. No food is wasted and the sight of people wasting it in restaurants is intolerable.

"I'm cold. In July I walk downtown on a sunny day with a shirt and sweater on to keep me warm. At night my well-fed room mate, who isn't in the experiment, sleeps on top of his sheets but I crawl under two blankets wondering why Don isn't freezing to death. My body flame is burning as low as possible to conserve precious fuel and still maintain life processes.

"I'm weak. I can walk miles at my own pace in order to satisfy laboratory

requirements but often I trip on cracks in the sidewalk. To open a heavy door it is necessary to brace myself and push or pull with all my might. I wouldn't think of trying to throw a baseball and I couldn't jump over a twelve-inch railing if I tried. This lack of strength is a great frustration. In fact it is often a greater frustration than the hunger. I eagerly look forward to the day when I can go upstairs two at a time or maybe run to catch a streetcar.

"And now I have edema. When I wake up in the morning my face is puffy on the side I was lying on. Sometimes my ankles swell and my knees are puffy, but my edema isn't as bad as that of several others whose flesh bulges out over their shoes in the evening.

"Social graces, interests, spontaneous activity and responsibility take second place to concerns of food. I lick my plate unashamedly at each meal even when guests are present. I don't like to sit near guests, for then it is necessary to entertain and talk with them. That takes too much energy and destroys some of the enjoyment that comes from my food. I no longer have that ardent desire to help millions of starving people; rather I feel akin to them and hope that I as well as they will benefit from scientific refeeding.

"I am one of about three or four who still go out with girls. I fell in love with a girl during the control period but I see her only occasionally now. It's almost too much trouble to see her even if she visits me at the Lab. It requires effort to hold her hand. Entertainment must be tame. If we see a show, the most interesting part of it is contained in scenes where people are eating. I couldn't laugh at the funniest picture in the world, and love scenes are completely dull.

"I can talk intellectually, my mental ability has not decreased, but my will to use the ability has. So my talk is of food and past memories, or future ambitions mostly in the cooking or eating line.

"That was starvation! Rehabilitation was carried on with the same food only more of it, but life came back slowly. The men were divided into four groups. I was in the lowest group and after 6 weeks of refeeding had gained one quarter of a pound! I felt better, however, as many pounds of edema had been replaced by healthy tissue. At the end of 6 weeks everyone was given an additional eight hundred calories daily. Now men in the lowest group were getting some 3000 calories (about the average daily amount of the American diet) but even on this diet the average weight gain in their group twelve weeks after the end of starvation was only 7½ pounds compared with nearly a 40 pound average loss.

"Now, eight months after the end of starvation, I am fat and healthy although my muscles have not yet returned to their former tone. I look back to those days in July and recall my feeling of apathy. Yet that was starvation *only*, for I had warm clothes and living quarters. I was free from disease and could keep clean; but I think of Europe and Asia and I see bodily discomfort increased multifold with the addition of disease and cold and filth. I see mental torture multiplied because of bitterness and sorrow from war's devastation, and the constant wonder if the day will ever come when food will again be plentiful."

Intellective Functions

ROGER BACON is credited with saying that "The brain is in some sort of custody of the stomach and relief of malnutrition gives relief of mental dullness" (J. Wilder, 1944). Feuerbach's dictum "Der Mensch ist was er isst" (Man is what he eats) may be regarded as another statement of the widely held belief in the intimate connection between man's physique, including the nutritional condition, and his intellect. This belief is not limited to philosophers. There is a distinct tendency on the part of some individuals professionally concerned with the problems of nutrition to overdramatize the importance of food in reference to mental capacity. Selling and Ferraro (1945), a psychiatrist and a dietitian respectively, without giving any figures concerning the frequency of the occurrence of such a phenomenon, have written: "If the practitioner [to whom the children are brought because they are not doing well in school] is well versed in dietetics and mental disturbances which may result from improper feeding, he will *often* [italics ours] recognize that the child's backwardness is the result of a vitamin deficiency, a mineral deficiency, or some other similar defect" (p. 16).

In famine literature one frequently finds loose statements concerning the deleterious effects of starvation on intelligence. For example, in the report on the 1945-46 nutritional survey of the internment camps in the Netherlands East Indies it was stated categorically: "No doubt in many cases decreases of intelligence were present" (Netherlands Red Cross Feeding Team, 1948, p. 48). Complaints of forgetfulness and incapacity to concentrate were registered; an apathetic disposition — sharply contrasting with occasional paroxysms of irritability — and a tendency toward sleepiness were also noted. For purposes of scientific analysis it is desirable to separate "mental capacity" and "mental activity." The former may be identified with the functional status of the cerebrum, especially the cortex. In view of the resistance of this organ to morphological changes induced by semi-starvation, one would expect the functional capacity to be maintained essentially undiminished. Operationally, we may define *mental capacity* as that level of intellective performance which is achieved when all factors, including motivation, but excepting the experimental variable (in this case the body weight), are at an optimum. Such conditions were closely approximated for testing intellective performance in the Minnesota subjects. *Mental activity*, especially in the sense of self-initiated mental work, is a more complex phenomenon, which need not bear a very close relationship to mental capacity. The latter is simply a limiting factor. It is intelligence in this sense of mental capacity,

as measured in terms of intellectual tests, with which we are concerned in this chapter.

Studies on Intelligence and Nutritional State

The relationships between intellect and physique, including the possible mental effects of malnutrition, were subjected to a critical analysis by Paterson (1930). Surprisingly enough, the experimental evidence is overwhelmingly negative. Using height and weight as well as anatomical and physiological age as criteria of physical development, the association between mental and physical status was found to be negligible.

Terman's (1926) study of gifted children might be interpreted as having yielded results that are contrary to this statement. As a group, the gifted children were physically superior to American-born children as a whole, deviating in the positive direction from the Baldwin standards in such characteristics as weight. They excelled in reference to local (California) norms as well. However, the correlations between mental age and physical traits — provided the chronological age was kept constant — were very low. Mental age and weight correlated to $+0.051$ in boys and $+0.035$ in girls, the total sample comprising 594 gifted children.

Numerous correlational studies on the relationship between anthropometric indexes of "nutritional status" and measured intelligence, carried out in the 1930s on "normal" populations, mostly school children and college students, indicated the presence of a positive, but extremely low, correlation between the two sets of variables. Heidbreder (1926) obtained correlation coefficients of $+0.03$ for 500 male freshmen and $+0.04$ for 500 female freshmen between the weight/height ratio and the score on the Minnesota College Ability Tests; neither value was significantly different from zero. Further relevant information is provided by contrasting groups of undernourished and normally nourished individuals. These studies, in order to yield valid information, must satisfy rigid requirements of stratified sampling and in this way eliminate the effect of factors which tend to be associated with measured intelligence, such as economic status.

An extensive field study on the mental effects of malnutrition was made by Blanton (1919). The subjects were children, between 6 and 14 years of age, in the public schools of Trier in western Germany. It was estimated that at least 40 per cent of these children had suffered from malnutrition for 3 years, but no data on changes in weight were given. One criterion of mental retardation was the percentage of children failing to pass their grade; whereas in the prewar years this figure was 8 per cent, it was 15 per cent in 1918 and 14 per cent in 1919. But the validity of this criterion is highly questionable. The author himself ascribed to malnutrition only a part of the increase in the percentage of failures, the remainder being due to the disorganization of schools and to other effects of war. The city was near the front, and the passing of troops to the front and of wounded from the front created an atmosphere of unrest. The schools often served as barracks, which resulted in crowded classrooms and shortened school hours. Home discipline suffered because of the absence of the fathers; this was reflected in the neglect of homework. The town was bombed 22 times and the

alarm was sounded nearly 100 times, frequently during school hours, necessitating the interruption of schoolwork to seek shelter in the cellar.

When the teachers were asked to compare their classes with those of prewar years, the level of work done previously by the upper 20 per cent of the classes was said to have been reached in 1919 by less than 10 per cent of the pupils. On the other end of the distribution, the prewar performance of the lowest 20 per cent was now considered the characteristic level of some 30 per cent of the students. Blanton was tempted to infer that 10 per cent of the children of superior intelligence had dropped down to the level of those of average intelligence and that 10 per cent of those who would have been previously rated as normal fell into the "dull" category. But when a German translation of the Stanford (Terman) revision of the Binet-Simon scale for measuring general intelligence was applied to a sample of 30 children suffering from malnutrition, no impairment of intelligence could be demonstrated. The distribution of the intelligence quotient did not deviate significantly from the pattern established for the general population: 23 children had I.Q.'s between 90 and 110, 3 above 110, and 4 below 90. In percentages, 76.7 per cent fell in the range of average intelligence, 10.0 per cent in the range of above average intelligence, and 13.3 per cent were classified as having a below average intelligence.

Blanton noted that all the children except the 4 having subnormal I.Q.'s showed a higher degree of intelligence than would be inferred from their schoolwork. The discrepancy was interpreted as an indication of a lack of "nervous energy"; the mental abilities were preserved but the endurance was low. In the morning when the children were fresh they were able to do the schoolwork normally. Later in the day tiredness set in and they were unable to carry on satisfactorily. The number of children considered "feeble-minded," including those who were placed in special schools, did not exceed the percentage to be expected in samples of the general population (less than 3 per cent). In summary, it seems that malnutrition did not result in a lowering of the level of general intelligence.

In an investigation carried out by Hunt, Johnson, and Lincoln (1921) on behalf of the Bureau of Educational Experiments in New York City, there was no significant difference in average general intelligence between the experimental group (underweight by more than 7 per cent according to height weight/age tables) and the control group. The I.Q.'s in the experimental group ranged from 64 to 126, in the control group from 55 to 125. Dowd (1922) compared the results of intelligence tests given to undernourished children enrolled in a nutrition class with those of another group of the same size ($N = 55$) selected at random from the population of the Bellevue Children's Clinic, and also with their own scores obtained on retesting after 6 months or more of nutritional care. Again, the distribution of the scores in the two groups followed essentially the same pattern, and the retest scores did not indicate improvement in mental level.

Z. A. Schumskaja reported on the mental effects of starvation and refeeding at a psychoneurological congress in Moscow in 1923; the data were summarized in a paper by Netschajeff (1927), who developed the battery of 10 mental tests

(repetition of digits and sentences, counting forward and backward, sentence completion, etc.) used by Schumskaja. The subjects were 98 children, both girls and boys, in the age range from 6 to 14 years, who had undergone starvation in the winter of 1922. By the time of the first examination 30 were considered to be heavier than "normal," 35 were classified as having a normal weight, and 33 were subnormal in weight. After about 3 months on a good diet the 3 groups gained 9.7, 12.7, and 17.8 per cent of the initially determined body weight. It is unfortunate that only these percentage increments, and not the initial values and absolute gains, were given. This is even more true with reference to the mental test results, which indicated increments of 22, 33, and 63 per cent, respectively; these can hardly be accepted at their face value.

The information on the effects of restriction of the mineral and vitamin dietary content, with particular reference to animal studies, was summarized by Fritz (1935). The effects of added thiamine on maze learning of the white rat were studied more recently by Lusk (1944) and Marx (1948). Lusk maintained 6 groups of growing rats on thiamine intakes varying from 2 to 15 micrograms per day. Over this range, no significant differences in maze learning were observed. The absolute levels in Lusk's experiment were low. In Marx's study, large supplementary doses (100 micrograms of thiamine hydrochloride) were given to rats maintained on the normal stock diet in an effort to provide a clear-cut test of the hypothesis that a liberal thiamine intake increases learning capacity. A multiple-T water maze was used, with 3 criteria of learning capacity: total number of errors, total time, and the number of trials preceding the mastery of the maze. None of the differences reached the level of statistical significance. Similarly, the results of a relearning test, which was run 2 weeks after the completion of the learning series, were negative.

We shall not attempt to summarize the often contradictory results of studies of the effect of supplementary feeding, including vitamin supplementation, on underweight children (e.g. Graper and Park, 1923; Smith and Field, 1926; Rosenberg, 1931; Kabanov and Marshak, 1934; Takasi, 1934; Seymour and Whitaker, 1938; Poull, 1938). For example, Rhoads *et al.* (1945) found in children no relationship between vitamin intake and intelligence, whereas Harrell (1943, 1946, 1947) reported positive effects from increased vitamin intake (for criticism of Harrell's studies, see *Nutrition Reviews*, 1946, 4:343-44). In the study reported by Robertson *et al.* (1947), using 36 pairs of twins and a large battery of psychological tests, no significant differentiation between the experimental (thiamine-supplemented) and the control subjects could be demonstrated.

The effects of a moderate but lengthy, and also of a severe but brief, deficiency of vitamins of the B complex on intellectual performance in normal young men were investigated by Guetzkow and Brožek (1947); the relevant literature was discussed. The experimental group was maintained for 161 days on a diet containing about one third the thiamine and riboflavin allowances recommended by the National Research Council and about two thirds the recommended amount of niacin; there was no indication of deterioration in intellectual performance or learning ability. An acute dietary deprivation of the B complex vitamins for 23 days resulted in a minimal deterioration in 2 out of 6 intellectual

tests in which speed appeared to be the essential factor; there was no evidence of an impaired rate of learning.

However, there is little doubt that definite, prolonged vitamin deficiencies, particularly when thiamine and niacin are involved, will eventually result in mental deterioration. Sebrell (1943) pointed out disturbances of memory, clouding of consciousness, and disorientation as symptoms occurring in the advanced stage of vitamin B complex deficiency. As far as we are aware, the deterioration and the mental effects of treatment were not studied quantitatively.

Experimental Studies on the Effects of Fasting and Semi-Starvation on Intellective Performance

The effects on intellective functions of total abstinence from food for a period of 24 to 72 hours were studied by Weygandt (1904). The perceptual speed, measured as the ability to read words exposed for a very brief period of time, was not affected. The character of verbal associations was explored both by the method of timed association reactions to 50 word stimuli and by the continued free association after a single stimulus word was given. The speed of the verbal reactions was not diminished, but there was some tendency to decrease the number of responses bound logically to the stimulus, such as those indicating cause-and-effect relationships. Productivity, expressed as the total number of freely associated words in 5 minutes, did not show consistent changes. Learning capacity, as judged from the increase in the test scores obtained during a series of daily testing trials, did not suffer from short fasts. For example, the total scores in a 5-minute addition test for subject A on successive days were 139, 168, 226, and 239; the second score in this series was obtained after a 24-hour fast. Performance in the addition test was unfavorably affected only when the fast extended beyond the first 24 hours. In subject W the scores were 255 and 265 on the two "control" days, 283 after 24 hours of fasting, 266 after 48 hours, and 226 after 72 hours; the scores obtained on the following 3 "normal" days were 341, 312, and 308, respectively.

Memorization of nonsense syllables seemed to be the function most sensitive to fasting; in subject W on 2 days preceding the fast the scores for a $\frac{1}{2}$ -hour test were 291 and 269; after 12 hours of fasting the score dropped to 208, and after 24 hours to 179; in 4 successive normal testing sessions the scores rose to 353, 483, 492, and 612. When the fast was repeated, the values after 12, 24, 36, and 48 hours of fasting were 395, 352, 288, and 229; with food, 335 and 432. It should be noted that the lack of proper controls and the general paucity of Weygandt's experimental material cause all inferences to rest on very tenuous ground.

During a 31-day complete fast (Langfeldt, in Benedict, 1915) a number of intellective functions were measured. Unfortunately there was no opportunity for pre-experiment practice, and the results in such tests as those of memory for words and digits and of cancellation speed, indicating an apparent improvement in intellective performance, should probably be interpreted simply as a result of continued practice. It may be noted that in the free association tests the time reaction to words designating foods was not different from that to other word stimuli. Inspection of the complete series of reaction words obtained on suc-

cessive days of the fast did not reveal any noticeable increase in the frequency of references to food.

A number of tests of intellectual performance were used by Marsh (1916) during a 1-week total fast: tests of association, color and form naming, continuous addition and subtraction (100 items in each list), and mental multiplication (10 2-digit problems). In the reproduction test the task was to recall the words constituting the word list of the preceding day. There was some decrement in the speed of intellectual performance but the mental deterioration appeared to be much less pronounced than the decline of grip strength. The fact that continued practice was superimposed on the effects of the fast complicates the picture and makes a definitive interpretation impossible. In the two tests of memory there appeared to be a decrement for the masculine subject and an increment for the feminine subject. Marsh interprets this finding in terms of sex differences; the data may be more correctly interpreted as indicating simply a chance fluctuation in these functions.

In the subjects studied by Miles (Benedict *et al.*, 1919) the gradual loss of 10 per cent of body weight had no significant influence on performance in mental tests. There was no change in memory for words, perceptual speed (number finding), or maze performance. In number cancellation, number addition, and a test involving a series of clerical tasks, the improvement in successive testing appeared somewhat slower in the undernourished group than in the control group, but the differences were negligible.

The test battery used by Glaze (1928) in his fasting experiments included 3 tests which may be classified as "mental": multiplication (2-digit by 2-digit numbers), color naming, and reading letters backward. The performance time in the multiplication test during a 10-day fast (subject A) and a 17-day fast (subject B) was within the range of normal variation. Subject C, who fasted 33 days, performed normally during the first half of the period; in the second half, after a transitory deterioration, his performance returned to a near-normal level. Results were similar in the color-naming test (12 cards with 100 colors each): there was no marked effect in subjects A and B, but a slowing down of the performance occurred in subject C (the time scores of this subject during the pre-fast, fast, and post-fast periods were 841 secs., 975 secs., and 742 secs.). The test of reading letters backward was taken only by subject A, who reacted more slowly during the fast, and by subject B, who showed no change.

Intellectual Functions in the Minnesota Experiment

As semi-starvation progressed in the Minnesota Experiment, complaints of inability to concentrate for any period of time and of difficulty in developing thoughts became numerous. By the end of the semi-starvation period a large proportion of the men felt that their judgment had been impaired. They reported further that their general alertness and comprehension had declined (see Chapter 38).

According to our clinical impressions, the intellectual capacity was essentially unchanged. Throughout the stress the subjects talked intelligently, though with decreasing speed, and appeared to think clearly. Memory disturbances and a

decrease in expressive power were only rarely encountered and did not seem to go beyond the range of normality.

Quantitative tests of intellectual performance were used to supplement the clinical judgment. The methods are described in the Appendix. Both speed tests, in which the working time was short and strictly limited, and power tests, in which no time limits were imposed and the working time was 8 to 10 hours, were used. The 6 speed tests were assembled in the Repeatable Test Battery; this battery was developed in the Laboratory on the basis of the factorial studies of L. L. and T. G. Thurstone (1941) and has been described in detail elsewhere (Guetzkow and Brožek, 1947). The 4 power tests (sentence completions, arithmetics, vocabulary, and directions) were those developed under E. L. Thorndike's direction at the Institute of Educational Research of Columbia University and assembled into a test battery designated by the initial letters of the tests, CAVD.

Repeated performance on a test leads to an improvement of the score; where we are concerned with possible changes in performance capacity, studied over a period of time, the factor of learning introduces unwelcome complications and has to be controlled. This can be done by the use of a strictly comparable control group, pre-experiment practice, or alternative tests. Although at the time of the semi-starvation experiment we had at the Laboratory another resident group,

TABLE 368

AVERAGE PERFORMANCE IN THE REPEATABLE TEST BATTERY OF INTELLECTIVE FUNCTIONS. All the tests were of short duration (maximum 3 minutes). The higher the scores, the better the performance. C = control; S12 and S24 = 12 and 24 weeks of semi-starvation; R9 = 9 weeks of rehabilitation. $N = 32$. (Minnesota Experiment.)

Test and Function	C	S12	S24	R9	S24 - C	$F_{S24 - C}$	R9 - C	$F_{R9 - C}$
Flags (perception of spatial relations)								
<i>M</i>	57.8	57.0	57.3	58.1	-0.5	0.30	+0.3	0.07
<i>SD</i>	12.9	11.9	12.8	11.7				
First letters (word fluency)								
<i>M</i>	45.9	47.5	47.2	46.2	+1.3	1.75	+0.3	0.14
<i>SD</i>	8.3	7.6	8.4	7.3				
Word-number recall (memory)								
<i>M</i>	27.1	28.1	28.2	28.8	+1.1	1.13	+1.7	2.47
<i>SD</i>	10.4	9.5	9.0	11.6				
Multiplication (number facility)								
<i>M</i>	43.4	41.9	43.1	42.9	-0.3	0.28	-0.5	0.90
<i>SD</i>	14.6	14.0	14.6	13.8				
Letter series (inductive reasoning)								
<i>M</i>	15.4	15.7	15.0	15.6	-0.4	0.55	+0.2	0.18
<i>SD</i>	4.6	4.0	4.6	4.1				
Number checking (perceptual speed)								
<i>M</i>	43.4	41.0	43.3	42.4	-0.1	0.01	-1.0	0.77
<i>SD</i>	11.6	10.9	11.9	12.4				

TABLE 369

AVERAGE PERFORMANCE IN THE CAVD TEST OF INTELLECTIVE FUNCTIONS. There were no time limits on the test; the average time taken to complete the test was about 8 hours. C = control; S12 and S24 = 12 and 24 weeks of semi-starvation.

$N = 32$. (Minnesota Experiment.)

	C	S12	S24	S12 - C		S24 - C	
				M	F	M	F
Total raw scores							
M	137.9	135.8	133.5	-2.1	1.16	-4.4	8.99[**]
SD	26.1	28.3	26.8				
"Altitude" scores							
M	426.2	424.7	421.7	-1.5	1.81	-4.5	28.93[**]
SD	11.1	12.2	9.0				
Raw scores in sub-tests							
Completions							
M	32.6	32.9	32.6	+0.3	0.17	0.0	0.00
SD	7.7	7.3	7.6				
Arithmetics							
M	39.9	36.7	35.9	-3.2	15.04[**]	-4.0	21.68[**]
SD	10.4	11.3	11.0				
Vocabulary							
M	29.8	32.1	32.1	+2.3	8.59[**]	+2.3	8.02[**]
SD	8.3	7.1	6.8				
Directions							
M	35.6	34.1	32.9	-1.5	1.93	-2.7	15.63[**]
SD	5.0	7.4	6.2				

which was tested by the same techniques, this group was small and the results obtained could be used as suggestive evidence but not as unquestionable control data. The semi-starvation experiment was so designed that each subject served as his own control. In the Repeatable Test Battery the factor of learning was controlled by intensive pre-experiment training to a plateau. In the CAVD the use of alternative forms of the test (form 2 at C, form 3 at S12, and form 4 at S24) and the span of time between the 3 testing sessions eliminated the factor of practice.

In the tests of the Repeatable Test Battery (see Table 368) the mean changes in starvation occurred in both the positive and the negative direction and were within limits of chance variation, as indicated by the F ratios. Because each subject acted as his own control and it was conceivable (but not probable) that the deteriorative effects of semi-starvation were masked by continued training, the tests were repeated at the same intervals of 3 to 4 weeks during the rehabilitation period. In no case did the rehabilitation values (R9) differ significantly from the pre-starvation means (C).

The results of the CAVD test of intellectual functions are presented in Table 369. The total raw scores for the 5 most difficult levels of the test (levels M, N, O, P, and Q) were slightly but consistently lower during semi-starvation. The changes were more impressive when the raw scores were translated into "altitude" scores (see the Appendix on methods). However, it should be pointed out that even these changes were minimal and do not represent an impairment that

would be detectable clinically. The raw scores obtained in the sub-tests reveal that the changes were far from uniform, the largest decrement occurring in the arithmetics test. In the vocabulary test there was a change in the opposite direction. In the completions test there was no change at any time. In view of these facts and the possible slight differences among the 3 alternative forms of the CAVD battery, the statistical significance of the over-all change should not be overemphasized.

In order to explore at least one facet of the capacity to learn, which is an important component of "intelligence," the test of crossing out 4s was administered in 18 1-minute trials, separated by 90-second pauses. There were two testing sessions, one toward the end of the semi-starvation period and the other, regarded as "control," after 3 months of rehabilitation. In this test learning results in an increase of the score. Although at R12 the group started from a higher initial level as a result of previous practice, the *rates of learning* on the two occasions (S24 and R12) were essentially identical (see Figure 113). This fits in with the other test results and with the clinical observations and increases our confidence that "intelligence" was not adversely affected by the reduction of food intake and the consecutive decrease in body weight.

In view of this cumulative evidence about the absence of a biologically significant deterioration of intellectual capacity during starvation, the subjects' estimates of their loss of intellectual abilities must be attributed to physical disability and emotional factors. It was the narrowing of interests, apathy, and lack of initiative in carrying on conversation and study which led the men to conclude that they had suffered actual loss in intellectual powers. Some subjects were able

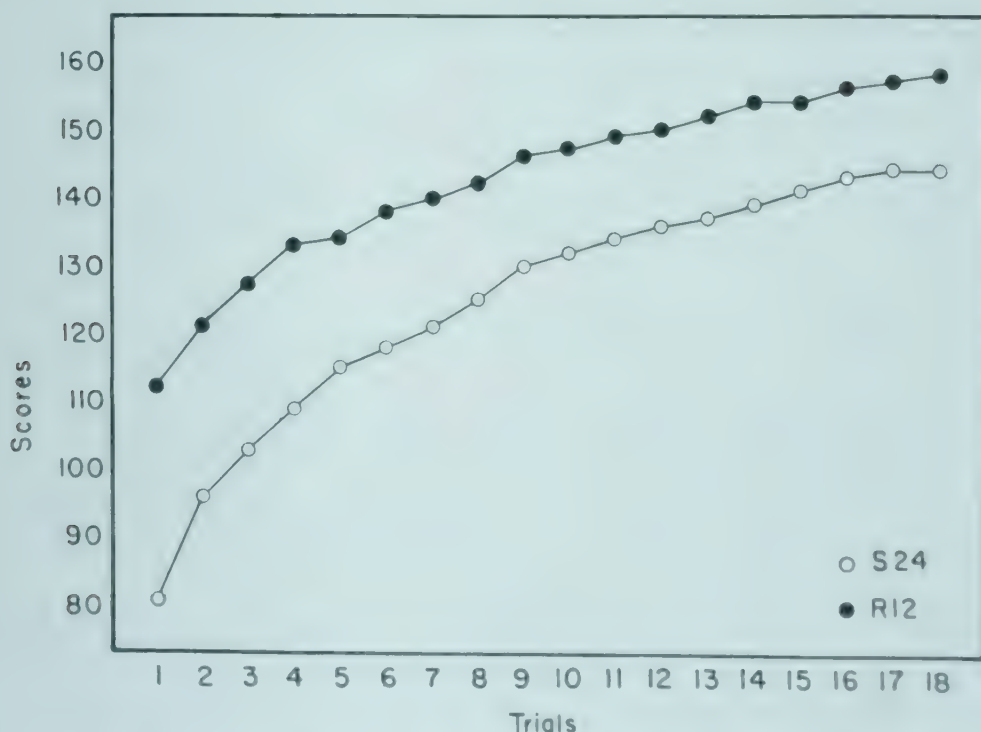


FIGURE 113. LEARNING IN THE TEST OF CROSSING OUT 4s. Single sessions, 18 trials. $N = 32$. (Minnesota Experiment.)

to carry a heavy academic load at the University throughout the semi-starvation period. However, the study program initiated by the group itself and designed to train the men for reconstruction service in Europe failed to hold their interest.

In rehabilitation there was a pronounced decline in the number of complaints about lack of alertness, inability to concentrate, poor comprehension, lack of judgment, and deterioration of memory. On the other hand, the performance in the Repeatable Test Battery did not change. Inefficiency and poor habits of study persisted. By and large, up to R12 the men did not resume their academic studies and intellectual pursuits with the application and vigor that had characterized their efforts during the control period.

In summary, the measured intellectual performance did not change importantly in either starvation or rehabilitation. The complaints about intellectual inefficiency rose in starvation and declined during the rehabilitation period. Spontaneous mental effort and achievement declined during starvation, remained at a low level during the early phase of rehabilitation (R1 to R12), and only gradually returned to "normal."

Personality

AS FAR as we are aware, until now no psychometric studies of the effects of prolonged caloric deficiency on personality have been made under either field or experimental conditions. Even in the area of vitamin deficiencies, such as those involving vitamins of the B complex, in which changes in the feeling of well-being and in personality are pronounced (see e.g. Spies *et al.*, 1943), standardized ratings and personality tests have been used only recently (Brožek, Guetzkow, and Keys, 1946; Henderson *et al.*, 1947).

Changes in Semi-Starvation—Inventories of Guilford and Guilford-Martin

Changes in some of the traits constituting "temperament" were studied in the Minnesota Experiment by means of two inventories devised by Guilford (1940) and Guilford and Martin (1943). The traits were defined on the basis of statistical analysis of personality questionnaires. The aim of the factor analysis preceding the construction of the scoring keys for the final form of the inventory was to isolate unitary, "independent" variables of temperament. The traits were given psychological meaning and names on the basis of the inspection of items constituting the various scales. The authors refer to the traits by letters to indicate the special nature of the operations by which they were defined. The verbal labels are apt to be misleading and tend to identify traits measured by given sets of questions in this inventory with concepts having traditional clinical connotations.

The factors STDRC were defined as follows: S = social introversion, tendency to withdraw from social contacts; T = thinking introversion, inclination to philosophize, tendency to analyze oneself or others; D = depression, including feelings of unworthiness and guilt; C = cycloid tendencies, fluctuations in mood; R = rathymia, carefree disposition, liveliness and impulsiveness.

The factors GÁMIN were defined in the following terms: G = general pressure for overt activity; A = ascendancy in social situations as opposed to submissiveness, leadership qualities; M = masculinity of attitudes and interests, I = lack of inferiority feelings, self-confidence; N = freedom from nervous tenseness and irritability.

The direction of the changes resulting from semi-starvation coincided with that expected on the basis of clinical observations. The mean raw scores and the F-tests indicating the statistical significance of the difference between C and S24 values are given in Tables 370 and 371. There was a statistically highly signifi-

cant *increase* in scores on the scales of social introversion, depression, and cycloid tendencies (emotional instability) and a *decrease* in those on the scales of ascendancy (social leadership), self-confidence, and freedom from nervous tenseness. The decrease in the scores on the rathymia scale and in the general activity scores reached a 5 per cent level of statistical significance. The increase in thinking introversion was not significant, and the mean score on the masculinity-femininity scale remained unchanged.

TABLE 370

SCORES IN GUILFORD'S INVENTORY OF PERSONALITY TRAITS STDCR as applied in the Minnesota Experiment. C = control; S24 = 24 weeks of semi-starvation. N = 32.

Trait	C		S24		S24 - C	
	M	SD	M	SD	M	F
S (social introversion)	17.5	9.3	23.0	10.0	+5.5	19.50[**]
T (thinking introversion)	35.8	7.5	37.3	4.6	+1.5	1.55
D (depression)	12.9	9.3	19.4	9.1	+6.5	20.11[**]
C (cycloid tendencies)	17.6	9.7	23.9	9.7	+6.3	19.71[**]
R (rathymia, an uninhibited disposition)	36.3	9.9	35.1	7.7	-1.2	7.10[*]

TABLE 371

SCORES IN GUILFORD-MARTIN'S INVENTORY OF PERSONALITY TRAITS GAMIN as applied in the Minnesota Experiment. C = control; S24 = 24 weeks of semi-starvation. N = 32.

Trait	C		S24		S24 - C	
	M	SD	M	SD	M	F
G (general activity)	21.9	9.0	18.7	7.9	-3.2	4.75[*]
A (ascendancy, social leadership)	50.6	16.5	44.9	15.4	-6.7	9.23[**]
M (masculinity)	56.4	14.9	56.4	13.9	0.0	0.0
I (self-confidence, lack of inferiority feelings)	87.5	23.0	76.8	13.9	-10.7	16.48[**]
N (freedom from nervousness) . .	89.0	24.0	72.5	21.3	-16.5	53.86[**]

In the attempt to evaluate the "absolute" magnitude of the changes in the personality traits measured by the inventories of Guilford and Guilford-Martin, the mean values presented in Table 370 were expressed in reference to centile norms based upon 388 students at the University of Nebraska (see Figure 114). A centile expresses the percentage of men in the normative group who fell below a given raw score, and the use of centile scores makes it possible to project the mean changes against the distribution of the scores in the normative population. The data in Table 371 were expressed in terms of the so-called C-scores in Figure 115; for the interpretation of C-scores see the Appendix on methods.

On the basis of clinical observations and some of the other psychometric instruments used, one would expect changes of considerably greater magnitude than are indicated in Figures 114 and 115. The apparent lack of sensitivity of these inventories to severe, prolonged experimental stress, resulting in pro-

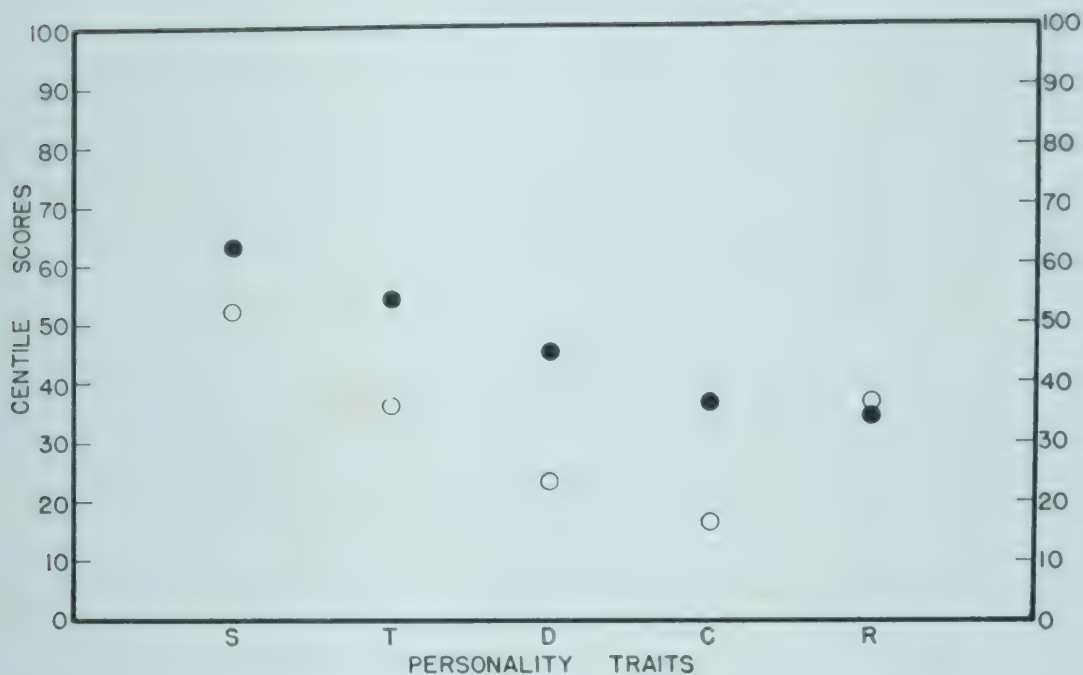


FIGURE 114. TRAITS MEASURED BY THE INVENTORY OF FACTORS STDCR. Open circles represent pre-starvation (control) data; solid circles indicate the values obtained after 6 months of semi-starvation (S24).
 $N = 32$. (Minnesota Experiment.)

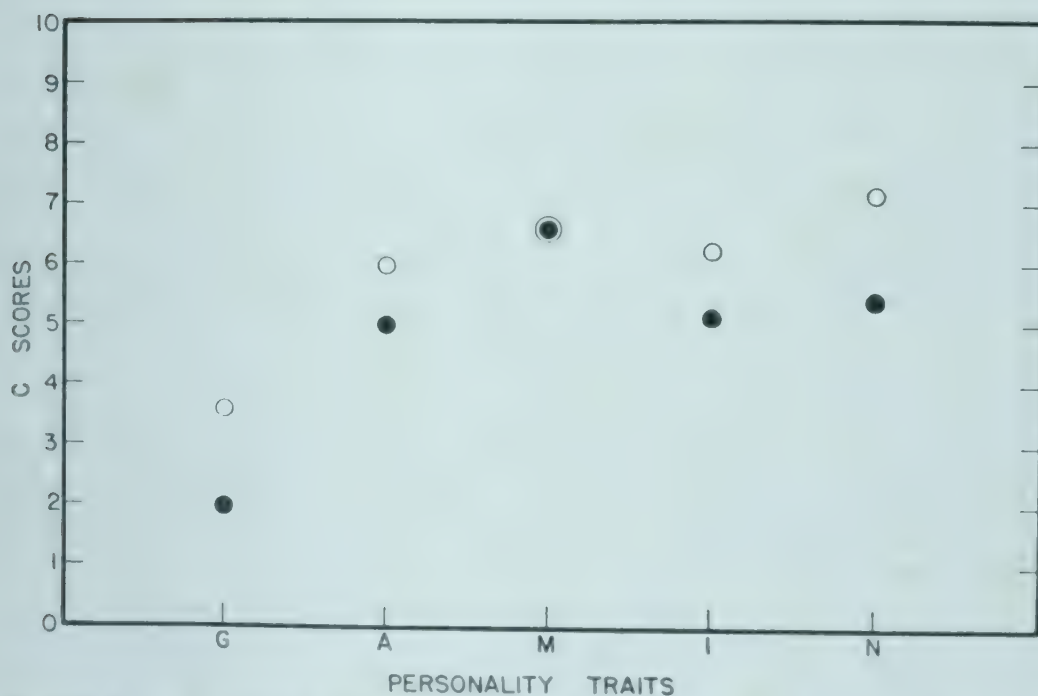


FIGURE 115. TRAITS MEASURED BY THE INVENTORY OF FACTORS GAMIN. Open circles represent pre-starvation (control) data; solid circles indicate the values obtained after 6 months of semi-starvation (S24).
 $N = 32$. (Minnesota Experiment.)

nounced changes in behavior, is strikingly demonstrated by the Depression scores. Although in this trait the change from C to S24 was statistically highly significant, the centile score of 45 at S24 would indicate that even at the height of semi-starvation the group score was well within the "normal" range. The study of the relative shifts in the scores, evaluated in reference to the consistency in responses of the individuals comprising the sample and expressed in statistical tests of significance, is in this case more rewarding than the use of "norms."

Changes in Semi-Starvation — Minnesota Multiphasic Personality Inventory (MMPI)

The technique used by Hathaway and McKinley (1943) for deriving the scales of the Minnesota Multiphasic Personality Inventory was diametrically opposite to that used for the construction of the inventories by Guilford and Martin, except that both started with a large pool of questions related to mental health and socioemotional adjustment. The starting point in the development of the MMPI was the administration of the inventory to patients with reasonably clear-cut psychiatric syndromes (Hypochondriasis = Hs, Depression = D, Hysteria = Hy, Psychopathic deviation = Pd, Paranoia = Pa, Psychasthenia = Pt, Schizophrenia = Sc, and Hypomania = Ma); the discriminatory items were selected by contrasting the responses of the patients with those obtained for the "normal" populations. (With reference to the clinical validity of the MMPI scores cf. Modlin, 1947.)

In addition to the clinical scales, the MMPI is provided with a key measuring the masculinity-femininity of interests, and with three scales designed as checks on the validity of the instrument in the particular instance of administration. The Question score (?) refers to the number of items concerning which the respondent was undecided; if many items fall into this category, the scales tend to lose their discriminatory sensitivity. The Lie score (L) indicates whether the subject is attempting to draw a "socially acceptable" picture of himself; this tendency, if present in a marked degree, would also distort the portrait obtained on the basis of the clinical scales. The F score was derived for a set of 64 items which were answered in the infrequent direction by less than 10 per cent of the normal subjects. A high F score may result from lack of comprehension and carelessness on the part of the subject or from clerical errors in recording the responses; high F scores will occasionally be obtained validly, especially in cases with diffused psychopathological disturbances (Kazan and Sheinberg, 1945; Brožek and Schiele, 1948).

In the Minnesota Experiment the mean standard score on the Question scale was equal to 50 at all times, which coincides with the "normal average." The Lie score was also very close to this level, varying from 52.2 at C to 50.9 at S24. The F score indicated a very slight increase from C to S24, with values of 53.3 and 54.9, respectively, but this slight trend simply reflected some of the clinically confirmed aspects of personality deterioration. This fact will be brought out in greater detail in the case studies.

The MMPI was used in order to determine the degree of "normality" of the subjects at the start of the experiment and, later, to follow personality changes

at different stages of semi-starvation and rehabilitation. In the control period the average standard scores for all clinical scales were close to the "normal average" of 50, except for some elevation in the Hysteria scores. The apparent "femininity" of the sample, indicated by a high Mf score, did not seem to have any clinical significance and probably reflects the fact that the group had more interests in cultural activities than the population on which the inventory was standardized. We have consistently found such elevated Mf scores in testing other samples of the population from which our group was drawn.

The scores obtained for the eight clinical scales may be interpreted both individually and with reference to the interrelationships between the scores as expressed by the profile. The use of standard scores (normal average = 50, $SD = 10$) makes the various scales directly comparable. Dealing with each scale individually we may first ask about the magnitude of the mean semi-starvation changes as related to the consistency of the changes in individual subjects. The data in Table 372 indicate a highly significant rise in the scores on the Hypochondriasis, Depression, and Hysteria scales. As to the "psychotic" items, no change was observed in Psychopathic deviation, Paranoia, and Hypomania scores, whereas there was a statistically highly significant elevation on the Psychasthenia and Schizophrenia scores. The fact that the average score on the Pd scale was initially low and that there was little change during semi-starvation indicates the absence of a tendency to develop aggressive, antisocial reactions or "character neuroses." It is of interest that three of the four subjects who failed to complete the experiment (and were not included in the group profile) did show significant elevations in the Pd score. The Masculinity-femininity scores showed a slight but consistent and statistically significant decrease in the "femininity" of interests; the drop is consistent with a general clinical impression that the Mf score tends to decrease as neurotic reactions develop.

The second criterion consists in considering the shift in the scores with reference to the distribution of scores in the general population. On the basis of statistical considerations and of clinical experience, the border line between "normal" and "abnormal" is drawn at the level of the standard scores of 70 (mean

TABLE 372

SCORES IN THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY as applied in the Minnesota Experiment. C = control; S12 and S24 = 12 and 24 weeks of semi-starvation; R1, R6, and R12 = 1, 6, and 12 weeks of rehabilitation. $N = 32$.

Scale	C		S12	S24		S24 — C		R1	R6	R12
	M	SD	M	M	SD	M diff.	F	M	M	M
Hypochondriasis	45.7	3.4	58.2	63.0	6.7	+7.3	110.1**	57.1	55.8	54.1
Depression	54.2	6.5	64.8	73.9	12.1	+19.7	109.6**	69.8	66.6	65.7
Hysteria	59.0	6.1	65.8	70.0	8.2	+11.0	52.2**	66.6	66.5	64.9
Psychopathic deviation .	52.2	7.5	53.0	52.9	6.5	+0.7	0.3	51.8	53.1	53.0
Masculinity-femininity .	69.8	10.3	68.0	66.9	8.1	+2.9	6.4 ^[*]	67.4	68.2	66.9
Paranoia	53.5	5.0	53.4	54.1	5.9	+0.6	0.3	53.0	54.7	53.2
Psychasthenia	45.7	7.4	46.8	51.9	7.7	+6.2	22.1**	50.0	49.0	49.1
Schizophrenia	47.5	7.5	49.2	55.1	8.9	+7.6	13.2 ^[**]	52.6	50.4	48.5
Hypomania	51.0	7.1	50.2	51.4	8.7	+0.4	0.1	50.4	50.9	50.6

+ 2SD). This line was crossed only by the mean Depression score, and it was reached by the mean Hysteria score. On the basis of this criterion, at the end of semi-starvation all mean scores on the "psychotic" scales were well within the limits of normality.

The third approach is to consider the profiles, obtained by connecting the standard scores on the various scales, in the control period and the changes in the profiles at the different stages of the experiment. The composite MMPI profile for the 32 men who completed the entire experiment is given in Figure 116. The profile obtained during the control period is completely normal, except for the elevated Mf (Masculinity-femininity) score. In semi-starvation the striking feature is the general elevation of the "neurotic" end of the profile (scales Hs, D, and Hy). This graphic presentation of the personality changes helps the experimenter to go beyond the atomistic approach inherent in concern with the single scales. In accordance with the clinical observations, the nature of the personality changes resulting from semi-starvation may be regarded as a diffuse "psychoneurosis." The right side of the profile is only moderately elevated and the mean semi-starvation (S24) scores, if obtained for an individual patient, would be considered entirely normal; in most of the subjects there was no evidence of "psychotic" types of reaction, although these did appear in a few individuals who showed more severe or unusual symptomatology.

The standards for the inventory were derived from samples of psychiatrically

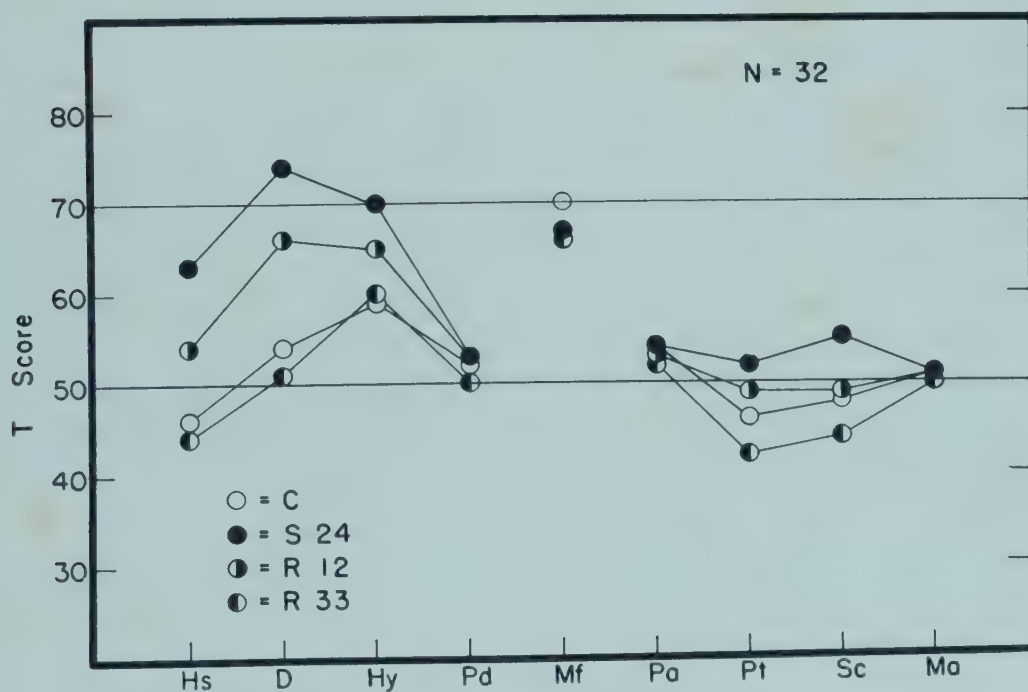


FIGURE 116. MINNESOTA MULTIPHASIC PERSONALITY INVENTORY, average values for 32 subjects who completed the experiment. The scores for individual subjects were obtained as the average of 2 administrations during the control period (C), at the end of the semi-starvation period (S24), and after 12 weeks of controlled rehabilitation (R12). The R33 values were obtained for 20 subjects during a follow-up examination made after 12 weeks of controlled rehabilitation plus 21 weeks of unrestricted diet. (Minnesota Experiment.)

normal subjects and of psychiatric patients, both of whom were considered to be *physically* normal and thus differed importantly from our semi-starved subjects. This difference posed the problem of whether the score elevations observed in the course of semi-starvation had approximately the same psychological significance they would have in physically normal subjects. This question was scrutinized in detail (Brožek and Erickson, 1948) by means of an analysis of the items constituting the three psychoneurotic scales of the inventory (Hypochondriasis, Depression, and Hysteria).

In evaluating each item, two frequencies (f and F) were taken into account: the number of men who answered a particular item in the "abnormal" direction in the control period (f) and at the end of 6 months of semi-starvation (F). The actual increase ($F - f$) in the number of men with abnormal responses was expressed as the percentage of the total *possible increase* ($N - f$, where N is the total number of subjects). This value was denoted as D , defined as

$$D = 100 \times \frac{(F - f)}{(N - f)}$$

A more or less arbitrary value of $D = 30$ per cent was chosen as indicating "significant" items, sensitive to the stress of semi-starvation. There were 29 such items out of a pool of 117 items constituting the three scales. Those items which deal with the "obvious" psychoneurotic symptoms and which were designated, for the purpose of the present analysis, as "somatic" and "psychic" are given in Table 373.

These items, and the psychic items in particular, indicate that important changes took place in the subjects' personality. In order to provide an empirical frame of reference for evaluation of the increase in the abnormal responses, the response frequencies of the semi-starvation subjects to the individual significant items were compared with frequencies obtained by Hathaway and McKinley for their psychoneurotic groups. Also, the responses of the experimental subjects during the control period were compared with Hathaway and McKinley's group of normal controls (cf. Brožek and Erickson, 1948). Although minor differences were present, owing in part directly to the condition under which the experiment was carried out, there was a striking similarity in the percentages of abnormal answers in the two comparisons. In the main the same items contributed to the elevation of the scores on the psychoneurotic scales in the semi-starved subjects and in patients with psychiatrically diagnosed hypochondriasis, depression, and hysteria but with no physical pathology. The item analysis documents in some detail the psychoneurotic character of the personality changes resulting from prolonged semi-starvation.

In rehabilitation all the semi-starvation changes were reversed, although the profile obtained at the end of controlled rehabilitation (R12) was still above the control profile. In this connection it may be pointed out that the first 12 weeks of rehabilitation were in reality a continuation of the stress. This was especially true of the first 6 weeks and of the men in the lower caloric groups. It was only some time after release from the controlled diet that complete rehabilitation was effected. Profiles on 20 subjects obtained after 33 weeks of refeeding had re-

turned to the "normal," pre-experiment level. Because the changes were produced by restriction of the diet and reversed by means of controlled nutritional rehabilitation, we may speak of an "experimental neurosis."

Dietary Factors in Personality Rehabilitation

We have emphasized the diagnostic importance of the *patterns of the scores* (the profiles) and interpreted the change in the group as a whole as a "neurosis" because of the significant elevation of the scores on the scales of the "neurotic triad." In evaluating statistically the differential effects of the dietary factors during the early phase of the rehabilitation period (R1 to R12), the scores obtained on the three scales (Hypochondriasis, Depression, and Hysteria) were considered separately. This procedure should not lead to thinking in terms of three crystallized and independent syndromes. A possible alternative would have been to average the three scores and carry out the statistical analyses on

TABLE 373

ITEMS ON THE PSYCHONEUROTIC SCALES which showed a marked increase from control to the twenty-fourth week of semi-starvation in the frequency of abnormal responses. $N = 32$. (Minnesota Experiment.)

	Percentage of Possible Increase (D)
Somatic Items	
I do not tire quickly. (False)	97
I am about as able to work as I ever was. (False)	94
I have never felt better in my life than I do now. (False)	92
I feel weak all over much of the time. (True)	88
I have had no difficulty in keeping my balance in walking. (False)	80
At times I am full of energy. (False)	62
Parts of my body often have feelings like burning, tingling, crawling, or "going to sleep." (True)	59
I seldom or never have dizzy spells. (False)	48
I am in just as good physical health as most of my friends. (False)	44
I wake up fresh and rested most mornings. (False)	42
I am neither gaining nor losing weight. (False)	38
I have had no difficulty in starting or holding my bowel movements. (False)	37
My hands and feet are usually warm enough. (False)	36
I hardly ever notice my heart pounding and I am seldom short of breath. (False) ...	33
Psychic Items	
My judgment is better than it ever was. (False)	85
I have difficulty in starting to do things. (True)	70
I dream frequently about things that are best kept to myself. (False)	67
I find it hard to keep my mind on a task or job. (True)	64
I certainly feel useless at times. (True)	48
I enjoy many different kinds of play and recreation. (False)	45
I have had periods of days, weeks, or months when I couldn't take care of things because I couldn't "get going." (True)	42
I like to flirt. (False)	37
I cannot understand what I read as well as I used to. (True)	32

TABLE 374

RECOVERY IN THE HYPOCHONDRIASIS SCORES OF THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY from R1 to R12 (Z = basal, L = $\frac{1}{2}$ 400, G = +800, T = +1200; U = protein-unsupplemented, Y = protein-supplemented; P = vitamin-unsupplemented, H = vitamin-supplemented). $dS24 = S24 - C$; $\Delta R = R - S24$. (Minnesota Experiment.)

Dietary Group	dS24	$\Delta R1$	$\Delta R6$	$\Delta R12$	$100 \times \Delta R12$
					dS24
Z	+14.0	-4.2	-4.8	-3.9	35.9
L	+22.0	-5.5	-8.2	-8.9	40.4
G	+16.7	-7.6	-9.5	-12.4	74.2
T	+16.4	-6.2	-6.5	-10.5	64.0
Z + L	+18.0	-4.9	-6.5	-6.4	35.6
G + T	+16.5	-6.9	-8.0	-11.4	69.1
U	+15.4	-4.6	-6.8	-7.2	46.8
Y	+19.2	-7.2	-7.8	-10.6	55.2
P	+17.2	-3.5	-6.1	-8.2	47.7
H	+17.4	-8.3	-8.4	-9.6	55.2

TABLE 375

RECOVERY IN THE DEPRESSION SCORES OF THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY from R1 to R12 (see the caption to Table 374 for the identification of the dietary groups) (Minnesota Experiment).

Dietary Group	dS24	$\Delta R1$	$\Delta R6$	$\Delta R12$	$100 \times \Delta R12$
					dS24
Z	+22.8	-4.2	-4.6	-2.2	9.6
L	+19.9	-6.2	-8.2	-8.8	44.2
G	+19.1	-4.2	-7.8	-9.8	51.3
T	+16.8	-1.8	-8.4	-12.1	72.0
Z + L	+21.3	-5.2	-6.4	-5.5	25.8
G + T	+17.9	-3.0	-8.1	-10.9	60.9
U	+17.0	-2.8	-3.7	-4.5	26.5
Y	+22.2	-5.4	-10.9	-11.9	54.1
P	+19.0	-4.9	-5.2	-8.3	43.7
H	+20.2	-3.3	-9.4	-8.1	40.1

this "neurotic" score. Being derived largely from different sets of responses, the three scores might be regarded as a triplication of the rehabilitation phase of the experiment and should provide us with a broader basis for drawing conclusions concerning the effect of dietary factors on recovery in the "neurotic" components of semi-starvation deterioration, provided the patterns of changes on the three scales are similar.

The rates of recovery in the Hypochondriasis, Depression, and Hysteria scores for the various diet groups are presented in Tables 374, 375, and 376. The amount of recovery at R12 in all three sets of scores tends to parallel closely the caloric intake. The differentiation is particularly striking for the Depression scale (see Figure 117). The recovery obtained at the end of the first week of nutri-

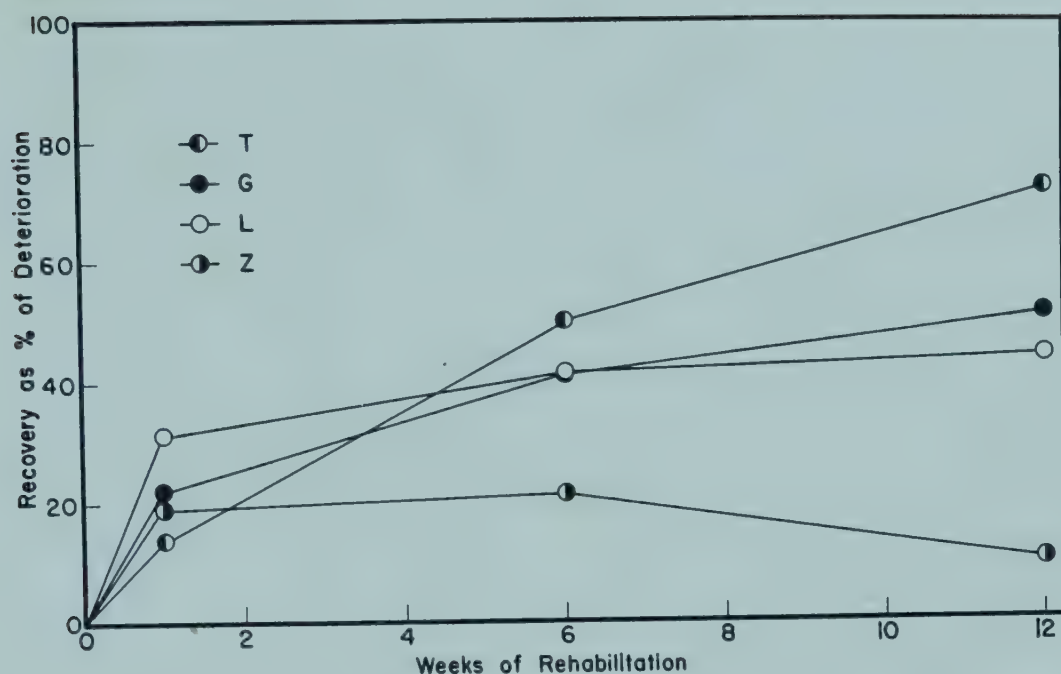


FIGURE 117. RECOVERY FROM R1 TO R12 IN THE DEPRESSION SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY IN THE 4 CALORIC GROUPS (Z = basal, L = +400, G = +800, T = +1200) (Minnesota Experiment).

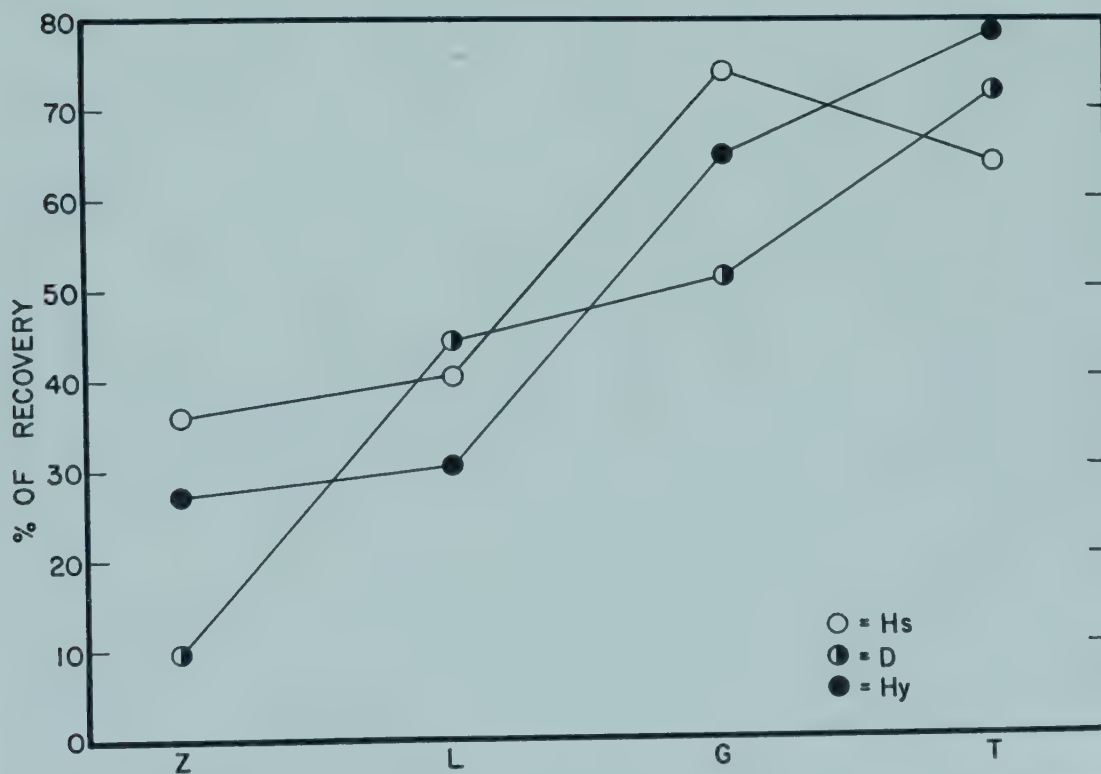


FIGURE 118. REHABILITATION CHANGES IN THE HYPOCHONDRIASIS, DEPRESSION, AND HYSTERIA SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY IN REFERENCE TO THE CALORIC LEVEL. The amount of improvement at 12 weeks of rehabilitation was expressed in terms of the deterioration suffered in semi-starvation.

In each of the caloric groups (Z, L, G, and T) there were 8 subjects. (Minnesota Experiment.)

TABLE 376

RECOVERY IN THE HYSTERIA SCORES OF THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY from R1 to R12 (see the caption to Table 374 for the identification of the dietary groups) (Minnesota Experiment).

Dietary Group	dS24	$\Delta R1$	$\Delta R6$	$\Delta R12$	$100 \times \Delta R12$
					dS24
Z	+11.8	-2.9	-1.1	-3.2	27.1
L	+14.8	-3.2	-3.5	-4.5	30.4
G	+9.4	-2.1	-4.0	-6.1	64.9
T	+8.2	-5.4	-5.4	-6.5	79.3
Z + L	+13.2	-3.0	-2.3	-3.9	29.5
G + T	+8.8	-3.7	-4.7	-6.3	71.6
U	+10.1	-3.2	-2.9	-3.6	35.6
Y	+11.9	-3.5	-4.1	-6.6	55.5
P	+11.9	-3.0	-3.1	-4.8	40.3
H	+10.2	-3.7	-4.9	-5.4	52.9

tional rehabilitation reflects the “lift” the subjects experienced at the termination of the semi-starvation regimen and their anticipation of improvement in the feeling of well-being, rather than any real physiological rehabilitation. At that time the amount of “recovery” was not related to caloric supplementation. By the sixth week of rehabilitation the picture became clearer, and by R12 the amount of recovery went hand in hand with the caloric levels. In the lowest caloric group the mean rehabilitation score at R12 was actually lower than that obtained in the first week of the rehabilitation period.

In Figure 118 the percentages of recovery were plotted against caloric levels for the scores of the “neurotic triad”: Hypochondriasis, Depression, and Hysteria. The tendency for a differential recovery in the caloric groups is evident. The regressions can be considered linear.

When the mean recovery scores are compared, taking only two caloric levels at a time, there is a significant difference in Hypochondriasis (Table 377) between the Z and G groups, between the Z and T groups, and also between the combined two lower (Z and L) and two upper (G and T) caloric groups. In the Depression and Hysteria scores no such statistically significant differentiation was obtained (see Tables 377, 378, and 379). The error variance in the Depression scores was very large.

The differences between the rates of recovery for the men who did and those who did not receive supplementary proteins or vitamins did not reach the levels of statistical significance for any of the three scales, except for the U vs. Y comparison of the recovery scores in Depression (Table 378); this was in part an artifact, since the two groups suffered a different amount of deterioration, reflected in the increase of the depression score by 17 and 22 points in the U and Y groups, respectively; one would expect the absolute rates of recovery to be proportional to the starvation changes. This is one of the instances in which a co-variance analysis would have increased the precision of discriminating between significant and non-significant differences in recovery.

TABLE 377

SIGNIFICANCE OF THE DIFFERENCES BETWEEN VARIOUS DIETARY GROUPINGS IN RECOVERY IN THE HYPOCHONDRIASIS SCORES OF THE MMPI at the end of 12 weeks of rehabilitation. V_{rep} (replicate variance) = 31.5. (Minnesota Experiment.)

	V_{bGr}	F
Z vs. L	100.0	
Z vs. G	289.0	9.12[**]
Z vs. T	175.6	5.57[*]
L vs. G	49.0	
L vs. T	10.6	
G vs. T	14.1	
(Z + L) vs. (G + T)	205.0	6.51[*]
U vs. Y	87.8	
P vs. H	16.5	

TABLE 378

SIGNIFICANCE OF THE DIFFERENCES BETWEEN VARIOUS DIETARY GROUPINGS IN RECOVERY IN THE DEPRESSION SCORES OF THE MMPI at the end of 12 weeks of rehabilitation. V_{rep} (replicate variance) = 87.7. (Minnesota Experiment.)

	V_{bGr}	F
Z vs. L	169.0	
Z vs. G	225.0	
Z vs. T	390.1	4.45
L vs. G	4.0	
L vs. T	45.6	
G vs. T	22.6	
(Z + L) vs. (G + T)	236.5	
U vs. Y	442.5	5.05[*]
P vs. H	0.3	

TABLE 379

SIGNIFICANCE OF THE DIFFERENCES BETWEEN VARIOUS DIETARY GROUPINGS IN RECOVERY IN THE HYSTERIA SCORES OF THE MMPI at the end of 12 weeks of rehabilitation. V_{rep} (replicate variance) = 28.34. (Minnesota Experiment.)

	V_{bGr}
Z vs. L	6.25
Z vs. G	33.1
Z vs. T	42.2
L vs. G	10.6
L vs. T	16.0
G vs. T	0.6
(Z + L) vs. (G + T)	47.5
U vs. Y	69.0
P vs. H	3.8

In order to compare the average amount of rehabilitation in the U and Y and the P and H groups, the recovery scores ($\Delta R12$) were expressed as percentages of the semi-starvation change (dS24). In the second step, this percentage of recovery in the supplemented groups (Y, H) was related to the recovery in the unsupplemented groups (U, P). This ratio would be larger than 1.00 if the supplementation had a beneficial effect. There were indications that additional protein in the diet had such an effect. The superiority of the vitamin-supplemented group over the placebo group was both numerically less impressive and less consistent (see Table 380).

TABLE 380

RECOVERY DURING REHABILITATION ON PSYCHONEUROTIC SCALES OF THE MMPI, for the protein-supplemented (Y) and -unsupplemented (U) and vitamin-supplemented (H) and -unsupplemented (P) groups in the Minnesota Experiment. The recovery values are expressed as percentages of the degree of deterioration that occurred during semi-starvation. The recovery in the supplemented groups is compared with that in the unsupplemented groups (columns Y/U and H/P). R12 = 12 weeks of rehabilitation.

Scale	Recovery at R12					
	100 $\Delta R12/dS24$		Y	100 $\Delta R12/dS24$		H
	U	Y	U	P	H	P
Hypochondriasis	46.8	55.2	1.18	47.7	52.2	1.09
Depression	26.5	53.6	2.02	43.7	40.1	0.92
Hysteria	35.6	55.5	1.56	40.3	52.9	1.31

As indicated earlier, at the end of the semi-starvation period, in addition to the changes in scores on the scales of the "neurotic triad" (Hs, D, Hy), there was a statistically significant increase in the Psychasthenia (Pt) and Schizophrenia (Sc) scores. In rehabilitation there was no significant relationship between the dietary factors and the rate of recovery in these scores.

At S24 there was a bias in the amount of deterioration in the Pt (Psychasthenia) scores, the mean values of the differences ($d = S24 - C$) for the caloric groups Z, L, G, and T being +1.2, +7.8, +7.5, and +8.4, respectively; the small change in the Z group was due to one subject who showed a big change in the opposite direction. At R12 the recovery in the Pt scores was essentially complete in all but the Z group, which showed a further deterioration, the rehabilitation differences ($\Delta = R12 - S24$) for the 4 caloric groups being +0.6, -8.0, -7.8, and -8.0. There was a similar bias in reference to the 2 protein groups, the dS24 in the unsupplemented (U) and supplemented (Y) groups being +4.8 and +7.7; the $\Delta R12$ values were -3.0 and -8.6. A statistical analysis which would not take into account the differences in the absolute amount of starvation deterioration would tend to lead to spuriously significant F-tests.

In the Schizophrenia scores the situation was similar, the mean starvation increments in the caloric groups Z, L, G, and T being, respectively, +3.4, +8.2, +8.4, and +10.1, and the recovery (R12) decrements being -1.5, -6.8, -8.4,

and -9.5. The starvation increments for groups U and Y were +5.5 and +9.6 and the rehabilitation decrements were -4.4 and -8.7; on a percentage basis the rate of recovery in the two groups can be considered closely similar (80 per cent and 91 per cent). In the P and H groups the semi-starvation changes were +7.8 and +7.2 and the mean recovery values were -7.3 and -5.8, or a percentage recovery of 94 and 81 per cent, the vitamin-supplemented group being somewhat inferior.

Measured Personality Changes in Later Rehabilitation

The MMPI served to characterize the progress of psychological recovery also in the later stages of rehabilitation. Considering the means for the 12 men who stayed at the Laboratory for an additional 8 weeks, there was a further significant improvement of the scores on the Hypochondriasis and Depression scales (see Table 381). The trend was observed in other scales as well but was

TABLE 381

SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY AT R20 (20 weeks of rehabilitation) and at other periods used for comparison. N = 12.
(Minnesota Experiment.)

Scale	C	S24	R12	R20	(R20 - R12)		(R20 - C)	
					M	F	M	F
Hypochondriasis	44.8	58.5	50.8	43.6	-7.2	28.29[**]	-1.2	2.51
Depression	52.2	69.9	64.7	57.7	-7.0	5.98[*]	+5.5	3.44
Hysteria	59.9	69.6	64.1	62.2	-1.9	2.43	+2.3	1.07
Psychopathic deviation .	49.7	50.9	51.2	49.8	-1.4		+0.1	
Masculinity-femininity .	66.0	63.2	63.6	64.3	+0.7		-1.7	
Paranoia	53.1	53.2	53.5	53.5	0.0		+0.4	
Psychasthenia	42.9	48.2	43.2	41.2	-2.0	1.66	-1.7	2.35
Schizophrenia	44.2	51.6	44.9	43.4	-1.5	1.19	-0.8	0.30
Hypomania	49.8	51.6	48.7	49.9	+1.2		+0.1	

TABLE 382

SCORES ON SELECTED SCALES OF THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY AT R20 (20 weeks of rehabilitation) and at other periods used for comparison. There were 6 subjects in each of the 2 sub-groups, Z + L and G + T. (Minnesota Experiment.)

Scale	C	S24	R12	R20	(R20 - R12)		(R20 - C)	
					M	F	M	F
Hypochondriasis								
G + T	43.5	59.5	48.5	41.3	-7.2	22.38[**]	-2.2	5.25
Z + L	46.2	57.5	49.7	45.8	-7.2	9.02[**]	-0.3	0.07
(Z + L) vs. (G + T) . .						0.00		1.41
Depression								
G + T	52.2	71.0	61.2	53.7	-7.5	18.05[**]	+1.5	0.30
Z + L	52.3	68.8	68.2	61.7	-6.5	1.29	+9.3	3.64
(Z + L) vs. (G + T) . .						0.03		1.95
Hysteria								
G + T	58.8	66.2	60.3	56.0	-4.3	8.37[*]	-2.8	3.71
Z + L	61.0	73.0	67.8	68.5	+0.7	0.40	+7.5	5.87
(Z + L) vs. (G + T) . .						7.45[*]		9.09[*]

less marked. In comparison with the control data the mean R20 values showed in no case a statistically significant difference from the control values, although the Depression score was still elevated, on the average, by 5.5 points.

As will be recalled, the group staying on at the Laboratory was composed of an equal number of men drawn from the Z + L and the G + T groups. In Table 382 the means for these groupings are presented, together with the tests of statistical significance of changes in the means. The decrease in the Hypochondriasis scores in the two sub-groups continued beyond R12 and by R20 had resulted in an improvement of identical magnitude (7.2 points). The absolute change from R12 to R20 for the Depression scale was also of about the same magnitude for both groups, but by R20 the mean score of the Z + L group was still elevated as compared with the control level. In the Hysteria scores a similar trend was present; the absolute differences were smaller than in the Depression scores but the individual responses were more consistent and the comparison of the Z + L and G + T groups indicates a statistically significant difference with respect to both the (R20 — R12) and the (R20 — C) differences.

At R33 the mean scores of 20 men, retested 21 weeks after the end of controlled rehabilitation, had returned for all scales to a level slightly lower than the pre-starvation values (see Table 383). Although the differences between C and R33 were numerically small, they attained statistical significance for a number of scales. At this time most of the men were released from their Civilian Public Service assignments, and this feeling of freedom may be reflected in the lowering of the general level of the MMPI profile. Actually, we were impressed by the close correspondence of the two sets of "normal" scores, obtained at C and R33, at an interval of more than one year.

TABLE 383

SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY AT R33 (33 weeks of rehabilitation) and at other periods used for comparison. $N = 20$.
(Minnesota Experiment.)

Scale	C	S24	R12	R33	(R33 — C)	
					M	F
Hypochondriasis	46.4	63.0	54.0	44.0	-2.4	4.80[*]
Depression	52.9	71.8	64.0	51.0	-1.9	1.00
Hysteria	60.3	71.8	66.0	59.6	-0.7	0.23
Psychopathic deviation ...	52.9	53.4	53.4	49.8	-3.1	2.34
Masculinity-femininity	69.9	66.7	66.7	65.5	-4.4	11.34[**]
Paranoia	54.3	53.5	53.0	52.4	-1.9	1.19
Psychasthenia	46.0	50.8	45.2	41.8	-4.2	9.48[**]
Schizophrenia	48.3	55.4	48.8	44.4	-3.9	6.13[*]
Hypomania	52.0	51.9	51.4	50.2	-1.8	2.34

Comment

In the psychometric study of personality during the starvation and rehabilitation periods of the Minnesota Experiment two sets of inventories were used: (1) those designed by Guilford and Martin on the basis of factorial analysis.

and (2) the Minnesota Multiphasic Personality Inventory developed in a clinical, psychiatric frame of reference.

The two sets of inventories have only two scales in common: Depression and Masculinity-femininity (Mf) of interests. The Depression scores in the two scales changed in the same direction, but the changes, evaluated in terms of the respective norms, were much more marked in the MMPI and paralleled closely the clinical judgment as to the amount of personality deterioration. The Mf scores on the MMPI indicated a high degree of "femininity" of interests at the control period, with a very slight decrease during the period of the stress; this trend was not reversed during rehabilitation. The Guilford and Martin inventory indicated low "masculinity" and no change during semi-starvation.

The MMPI, although not designed for use in an experimental situation involving repeated administration, proved to be a very useful instrument. The original standardization and the norms for the inventories of Guilford and Martin do not appear adequate. However, by taking into account the tests of statistical significance (consistency) and the qualitative aspects (direction) of the semi-starvation changes, the application of these inventories made possible a fuller description of the psychological alterations resulting from prolonged severe caloric deficiency than would have been possible otherwise.

Rosenzweig's Picture-Frustration test, a free association test, and the Rorschach test were also used in the Minnesota Experiment. The Picture-Frustration test (Rosenzweig, 1945) was administered at the end of starvation and after 12 weeks of rehabilitation; no significant changes were obtained for any of the scoring categories (Franklin and Brožek, 1949). Similarly, the responses in the free association test during starvation did not show any significant deviation from the responses made by a control group of 31 men who served as overhead personnel in the semi-starvation project or as experimental subjects in another nutrition experiment. The Rorschach test is widely used for personality diagnosis. In our experiment the Rorschach results were essentially negative. This may be interpreted as an indication of the absence of psychotic changes and basic alterations in the personality structure. The finding coincides with the lack of clinically significant changes in the psychotic scales of the MMPI; the exceptions are discussed in Chapter 41. The data obtained in the free association and Rorschach tests will be discussed in detail in separate publications.

The absence of significant changes in the "projective" tests under conditions of a severe "stress" is of considerable interest from the point of view of methodology in personality investigations. The finding was rather unexpected since the results of previous explorations by Sanford (1936, 1937), Levine, Chein, and Murphy (1942), McClelland and Atkinson (1948), and Atkinson and McClelland (1948), carried out under very mild conditions, appeared promising.

Psychological Case Studies

IN ANY severe and prolonged stress situation involving a large group of subjects a certain percentage of frankly psychopathological responses is to be expected. Although many of the subjects in the Minnesota Experiment had periods during which their distress was quite severe and all exhibited symptoms of "semi-starvation neurosis," there were only six men in whom the deterioration was striking (Schiele and Brožek, 1948). In four subjects (Nos. 234, 235, 232, and 233) the reaction to the semi-starvation regimen took the form of a "character neurosis," the men being unable to stay on the semi-starvation diet; in two of these subjects (Nos. 232 and 234) the response to the stress was particularly violent and bordered on a psychosis. One man (No. 20) developed a hysteroid reaction that led to self-mutilation. One (No. 5) exhibited neurological symptoms of probable hysterical origin. Subject No. 130 is of interest because he successfully completed the experiment in spite of a history of cyclothymic personality difficulties. Subject No. 2 made an optimal adjustment to the semi-starvation stress and is included in these case studies to provide a contrast to those who developed more severe symptoms.

The material for the case studies is based on direct observations of behavior during daily contacts with the subjects, information contained in the autobiographies and the diaries kept by the subjects, frequent interviews which were a part of the experimental routine, and detailed clinical examinations by Dr. Burtrum C. Schiele of the Department of Psychiatry, University of Minnesota. Out of the wealth of data obtained by the quantitative methods of observation, only the information provided by the Minnesota Multiphasic Personality Inventory (MMPI) will be included.

As indicated in the Appendix on methods and pointed out in Chapter 40, the MMPI scales were developed by contrasting the responses of patients exhibiting relatively well-defined psychiatric syndromes (Hypochondriasis = Hs, Depression = D, Hysteria = Hy, Psychopathic deviation = Pd, Paranoid reaction = Pa, Psychasthenia = Pt, Schizophrenia = Sc, Hypomania = Ma) with the responses obtained for a "normal" population. The masculinity-femininity of interests is measured by the Mf scale; the Mf scores were not incorporated in the personality profiles. It should be remembered that "normal average" scores for the MMPI scale are 50 and that the standard deviation in the normal reference population is equal to 10. The profiles illustrate graphically the personality patterns of the individuals, the changes that occurred in semi-starvation, and, in the subjects followed during the nutritional rehabilitation, the gradual return to

pre-starvation values. The profiles may be interpreted by considering a rise on the left side (Hs, D, and Hy) as indicating a "neurotic" type of reaction and a rise on the right side as indicating a more severe personality disorganization suggesting "psychotic" conditions.

In the preceding two chapters we have described the psychological manifestations of starvation found characteristically in all subjects: an intense preoccupation with thoughts of food, emotional changes with a tendency toward irritability and depression, decrease in self-initiated activity, loss of sexual drive, and social introversion. The composite MMPI profile for the 32 men who completed the entire experiment (Chapter 40, Figure 116) may serve as a general frame of reference for the individual profiles. Also the MMPI changes in subject No. 2 are characteristic of a "normal" reaction to semi-starvation (see Figure 120).

Subject No. 2

This subject is an example of the men who showed the least psychological deterioration.

He was a 24-year-old law student with strong interest and experience in the labor movement. The home provided adequate economic and emotional security, and the subject had a happy childhood. In college and law school he became a social and professional leader. He wrote a column in the college paper, organized a successful society, was its president for several years, and was active in campus politics. He was given a number of honors and repeatedly demonstrated his ability in many areas. This successful pattern continued in various Civilian Public Service assignments.

Neurotic traits in this individual were minimal. In his youth he had been shy and self-conscious with girls, was slow to learn to dance, and worried for some time over severe facial acne. The subject's sex drive was moderate. He had been interested in a number of girls but usually kept these relationships secondary to his work and professional ambitions. Observations during standardization confirmed the impression that the subject was capable, successful, and well integrated. He was adaptable, had a workable philosophy of life and definite goals, both immediate and future. He was well poised and was accepted by both the Staff and the members of the group.

He suffered a full share of the consequences of semi-starvation. His physical symptoms were of average severity except for a relatively large amount of edema in the last weeks of the semi-starvation period. His weight at the beginning of semi-starvation was 73.5 kg., at the end of semi-starvation 55.9 kg., with a low of 54.6 kg. 5 weeks earlier; the gain was due to accumulated edema (see Figure 119).

His psychological symptoms were likewise typical of semi-starvation. He was lethargic, mildly depressed, and somewhat irritable. The latter two symptoms were less marked in this subject than in many. He suffered from hunger pains and was preoccupied with thoughts of food as were all the other subjects, although he seldom talked about it. His sexual interests dropped off severely in the early part of semi-starvation. At the beginning of the experiment he became acquainted with a girl with whom he had intellectually much in common. He

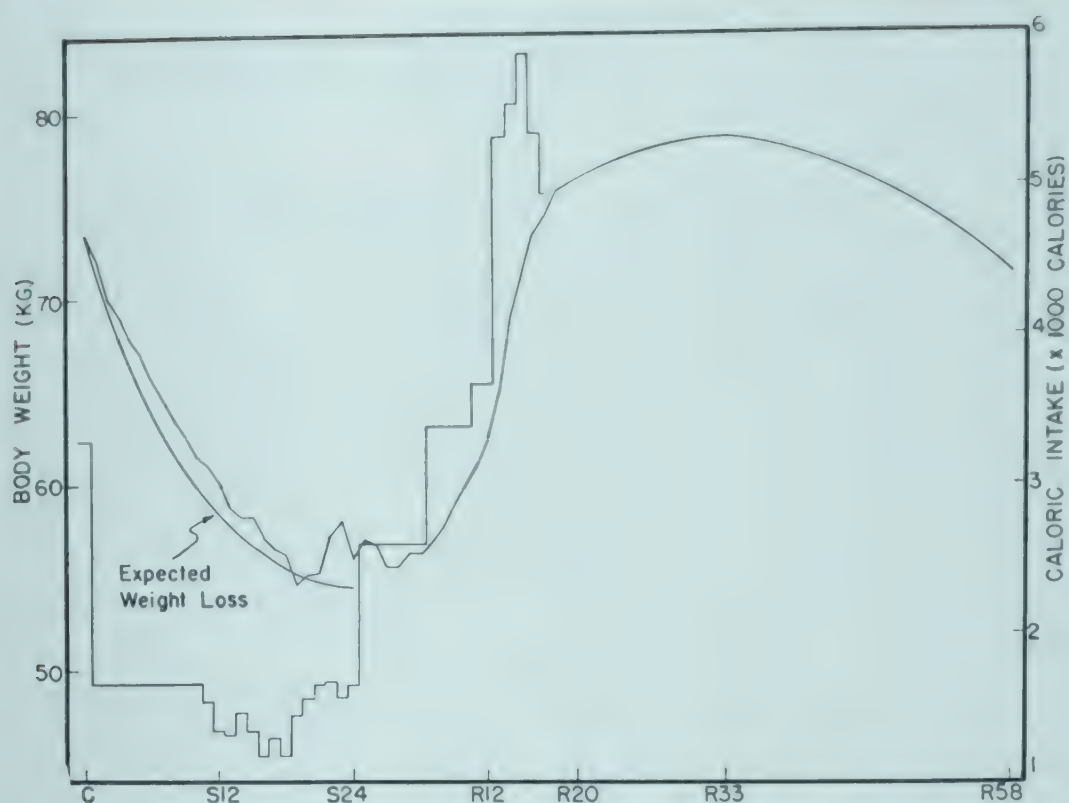


FIGURE 119. WEIGHT CHANGES AND CALORIC INTAKE FOR SUBJECT NO. 2 AT DIFFERENT PHASES OF THE MINNESOTA EXPERIMENT. In this and other figures of this chapter the "expected weight loss" line indicates the rate of loss of body weight predetermined for the subject at the start of the semi-starvation period. The continuous variable of body weight and the discontinuous variable of caloric intake are represented by the other 2 lines. C = control; S12 and S24 = 12 and 24 weeks of semi-starvation; R12, R20, R33, and R58 = 12, 20, 33, and 58 weeks of rehabilitation.

was greatly surprised to note the degree to which semi-starvation reduced his interest in this friendship.

On the positive side it may be said that he had little or no temptation to break the diet, and that he complained less than the average subject in spite of showing the same amount of physical deterioration. When he entered the experiment he set about completing the last semester of his law school studies. On obtaining the L.B. degree he began taking courses in political science, and completed most of the requirements toward an M.A. in that field. He was able to do this in spite of the distressing symptoms. He showed his maturity of judgment by anticipating that the early part of rehabilitation would be a continuation of the stress.

During the rehabilitation he was in the experimental group receiving a supplement of 800 Cal. In spite of this he felt little or no progress for the first 33 weeks of the rehabilitation period; actually his body weight decreased, owing to the loss of edema fluid, from an average of 56.9 kg. for the first week to an average of 55.5 kg. for the third week of rehabilitation. However, satisfactory completion of the starvation period did offer him some mental relief, and tangible evidences of recovery gradually became manifest.

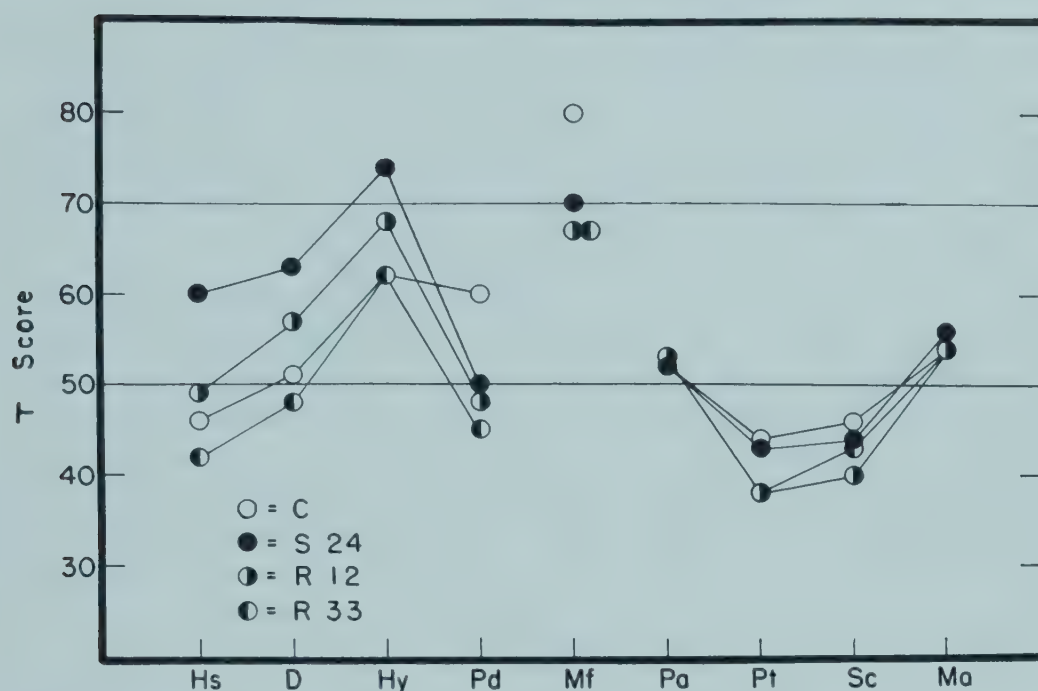


FIGURE 120. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT No. 2 during the control period (C), at the end of semi-starvation (S24), and after 12 and 33 weeks of rehabilitation (R12 and R33) (Minnesota Experiment).

The Minnesota Multiphasic Personality Inventory profiles for standardization and for R33 were normal (see Figure 120). At the end of semi-starvation the profile shows a moderate elevation of the "neurotic triad" (Hs, D, and Hy), the rise in the scores on all three scales being of about the same magnitude. The profile fits in with the clinical picture of a mild neurosis.

In summary, the psychological symptoms in this man were minimal yet definitely present. The experimental character of the semi-starvation neurosis was demonstrated by the fact that it developed as a result of the nutritional stress and disappeared on rehabilitation. This subject will serve as a contrast to those who had more severe and complex symptomatology.

Subject No. 234

This subject found himself unable to stay on the semi-starvation diet in spite of his strong desire to do so; the ensuing conflict precipitated a border-line psychotic episode which necessitated his removal from the experiment.

During standardization this 24-year-old man appeared to have everything needed to make his sailing smooth. He was charming, handsome, artistic, and had a "gift of gab." His standing in the group was high, and he taught with success a class in German which was a part of the educational program developed to prepare the men for relief service in Europe. He held a salaried position as a church organist while serving in the experiment. His obvious assets prompted the feeling that he was more mature than he actually was.

This man came from a comfortable urban home in which servants carried

the major burden of raising the six children. It appears that the subject had never been very close to his parents and that, particularly in his early youth, he was not well accepted by those of his own age. However, he enjoyed school, was a capable student, and obtained a university degree. Being talented musically, he planned to follow music as a vocation. Throughout life he had had more interest in boys than in girls and for several years he had carried on a "beautiful friendship" which gave evidence of a personality inversion.

The development of the unusual response to the experimental stress may be outlined as follows: In the first few weeks, while suffering the usual symptoms resulting from reduced food intake, he was troubled by strange dreams of "eating senile and insane people." His weight loss curve became aberrant as early as the third week of semi-starvation, which may indicate a nonadherence to the diet (see Figure 121). During the eighth week he flagrantly broke the dietary rules, eating several sundaes and malted milks; he even stole some penny candies. He promptly confessed the whole episode, became self-depreciatory, and felt he was not good enough to be retained in the experiment.

Judging from the content of his diary, the subject was clearly aware of the conflict between his desire to save face and his desire to leave. In an apparent attempt to strengthen himself he listed his reasons for sticking to the experiment: a desire for the personal satisfaction of completing it; the admonitions of his buddies; the approbation of his church, family, and friends; and the desire

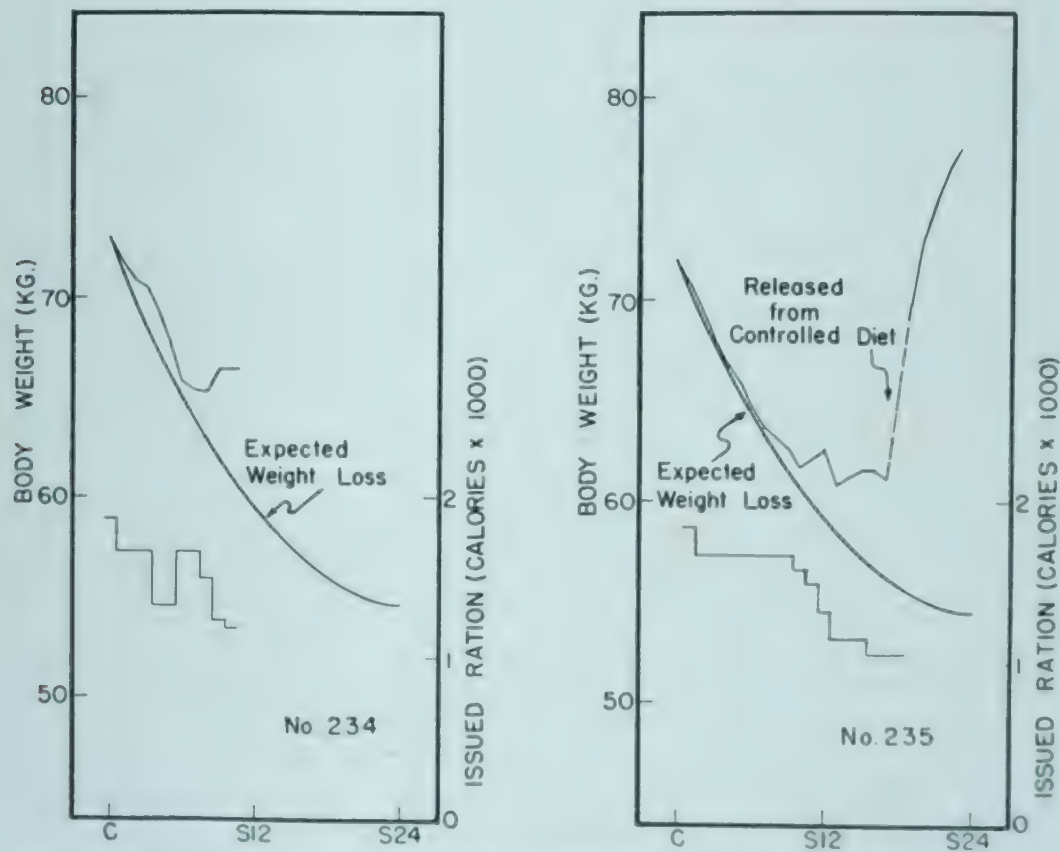


FIGURE 121. WEIGHT CHANGES AND CALORIC INTAKE FOR SUBJECT NOS. 234 AND 235 (see caption, Figure 119).

to do service to starving Europe and to uphold the ideals of the Civilian Public Service program. This may have been partially effective because his weight began to decrease more rapidly. His next diet violation was halfhearted: he stole and ate a few raw rutabagas (cooked ones being one of the main articles of the diet).

Early in the ninth week the conflict became more intense. He spoke of being unable to stop the whirling ideas going through his mind about "food, food, food" and began to display other signs of a serious personality disturbance. He became overwrought and began to write voluminously in his diary. His writing tended to ramble, with suggestions of a flight of ideas. He tried to turn to God in an attempt to solve his conflicts. For a few days he felt a new strength and even vowed to get a job in a grocery store to test himself. However, he developed insomnia and concluded, "I'm not the strong-willed egg that people seem to think I am." He went on a minor spree of shoplifting, stealing trinkets that had little or no intrinsic value for him; this may have served as food-substitutive behavior but did not provide real relief.

Since it became plain that he was unable to control himself, he attempted to save face with a number of rationalizations: he was an individualist and was not meant for regimentation; the whole experiment was a failure, and he should persuade the other men to quit with him; and finally, he had done a great deal for the group already and might as well quit. There were threads of self-depreciation and guilt, but these became less evident as time went on.

After discussions with the Laboratory Staff he once more attempted to get back in line; he volunteered to give up his money and checkbook, and he even asked for a "buddy" to supervise him constantly. When this failed utterly and it became necessary to place further restrictions upon him, he developed a violent emotional outburst with flight of ideas, weeping, talk of suicide, and threats of violence. Because of the alarming nature of his symptoms he was released from the experiment and admitted to the psychiatric ward of the University Hospitals.

At this time he presented the picture of a hypomanic: he was overly talkative, emotionally unstable, and somewhat elated. Within a few days' time, however, his symptoms subsided. Though he ate large amounts of food, he did not stuff himself to the point of becoming sick, and at the time of his release from the hospital he showed little gross evidence of personality disturbance.

This subject's MMPI (see Figure 122) taken during standardization is normal; the profile secured in the tenth week of semi-starvation (just before his release from the experiment) is clearly pathological. The elevation of the Pd scale and of the entire right end of the profile is indicative of a serious type of personality disorganization; these changes are in a marked contrast with those in subject No. 2. There was also a rise in the score on the F scale, from 50 in the control period to 73 in the tenth week of semi-starvation. In this instance the elevation cannot be interpreted in terms of F's being a "validation" scale but as an indication of severe personality disturbance.

In summary, the subject is a bisexual individual with poor personality integration and weak self-control, although he appears to have sufficient assets to adjust to the ordinary circumstances of life without much difficulty. He first

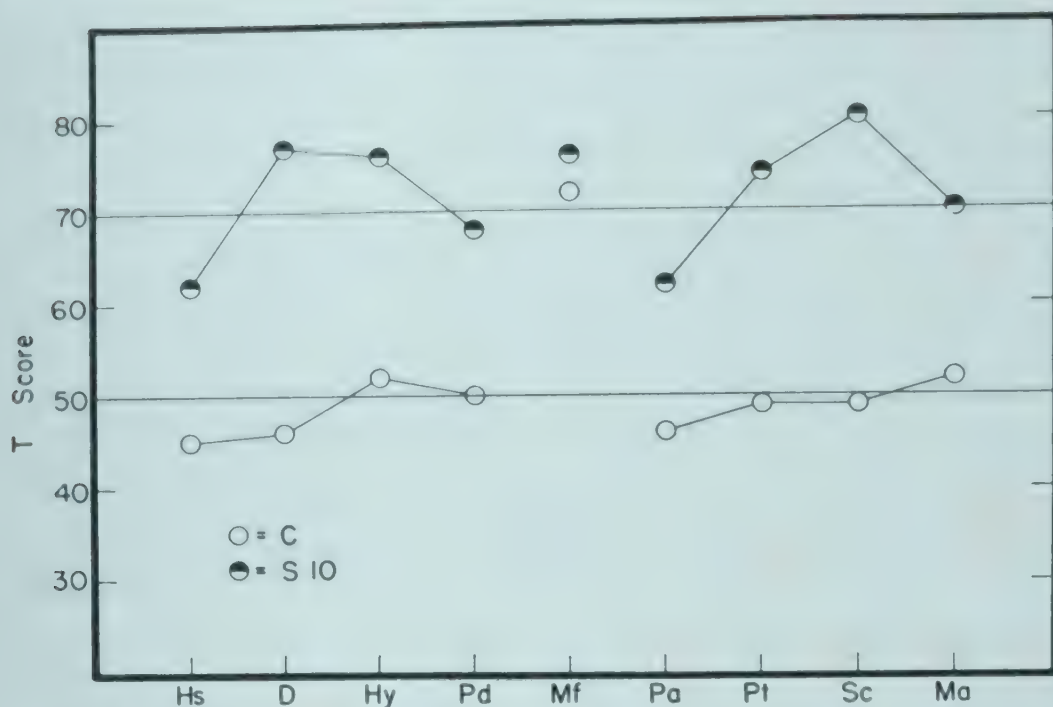


FIGURE 122. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT No. 234 during the control period (C) and after 10 weeks of semi-starvation (S10) (Minnesota Experiment).

slipped on impulse; this resulted in remorse and guilt, though he received some relief from confession. He tried to obtain support from his religious beliefs but slipped again. The mechanism of substitution, expressed in such acts as stealing trinkets, was ineffective. When threatened with forceful restrictions, he "blew up." This is an instance in which the experimental stress precipitated a reaction pattern that could be considered "psychotic." In spite of the short duration of the psychotic symptoms, the syndrome had many features of a manic psychosis. Many clinicians, on the other hand, would prefer to classify this disorder as a border-line psychotic episode in a "psychopathic" personality.

Subject No. 235

This 25-year-old was another who failed to adhere to the diet. He exhibited a mild clinical neurosis and in addition developed hematuria.

There seemed to be nothing unusual in his background. He and his seven siblings were raised on a farm and had a happy home life. After completing high school the subject worked for a time in an automobile factory and later attended college. He appeared to be a perfectly satisfactory member of the experimental group. He was well-met, friendly, and enthusiastic. He had entered the experiment because of a sincere and long-standing interest in nutrition and in foreign relief. He had definite plans for the future, intending to make rural cooperative farming his life work. He had a healthy interest in the opposite sex and during the experiment he corresponded regularly with the girl he hoped to marry. He was a hard worker and spent much of his free time as a bookkeeper in a cooperative grocery store.

On further observation, however, it became evident that the subject had hysteroid characteristics and other signs of a neurotic temperament. In interviews he was good-natured and easygoing but showed an immature "Pollyanna" attitude. He wrote copiously in his diary and ended every other sentence with an exclamation point. He always expressed great self-righteousness and optimism; this attitude is illustrated by his declared purpose in life: "To do that which is right in the sight of God our Maker, trying always to advance His Kingdom on earth. Truly, life is worthwhile!"

This subject's particular pattern of response began to show itself in the first few weeks of semi-starvation. Though he had no outward difficulty, he talked excessively about how well he was taking the stress. He emphasized that he suffered little or no food craving, had no temptations even when handling food in the grocery store; his adjustment to the routine was complete.

During the seventh week of the semi-starvation period he became unsettled and restless. One evening while working in the grocery store, he suffered a sudden "complete loss of will power" and ate several cookies, a sack of popcorn, and two overripe bananas before he could "regain control" of himself. He immediately suffered a severe emotional upset, with nausea, and upon returning to the Laboratory he vomited. He promptly made a complete confession to the Staff, trying at first to save face by referring to his loss of control as a "mental blackout."

In the next few days his pattern of defense began to reassert itself; he was "sure" that he had learned his lesson, that he would thereafter be alert to the possibility of temptation. A few days later he stated that he was "serene and secure" with a feeling of "complete control." The only evidence of his underlying uncertainty was found in his pressure to talk and write about his "little difficulty." On the surface all went well for the next few weeks. However, his weight failed to go down in spite of drastic cuts in the diet — a strong indication that he was again taking extra food (Figure 121). As this became evident, signs of a definite though mild clinical neurosis began to appear. The subject became increasingly restless and uneasy. He was self-depreciatory, expressing disgust and self-criticism. He made vague and ambiguous remarks about dietary irregularities, but was unable to face the issue squarely.

In mid-starvation he began to experience hematuria, a disorder which he had had some years previously; the urologist reported inflammatory changes of obscure etiology in the posterior urethra. Because of the discrepancies in the weight curve and because of the urological complications the subject was released from the starvation diet during the eighteenth week. He wrote later: "I received the news with mixed emotions, having sincerely desired to see the experiment through and yet realizing that my violations of diet would make results inaccurate! Sense of failure was almost predominant! Mentally, emotionally, and physically I was a mess. My first thought was escape from it all! Try and forget it as just a bad dream! The transfer to another unit didn't go through, and when asked to stay on till Oct. 20th and help out in the kitchen I felt honor bound to do just that and to do the best job I knew how. And again, it was no doubt the best thing that could have happened. Leaving here when I came off diet would have been purely an escape. Staying here, while difficult to adjust to, has helped

me to recover the assurance, the interest in others, and the will to go forward, that has always driven me. While sincerely regretting my failures here, it [the experiment] has taught me a lesson in humility and understanding that will be priceless to me. And perhaps in other areas I can, with God's help, make my contribution to the society in which I live."

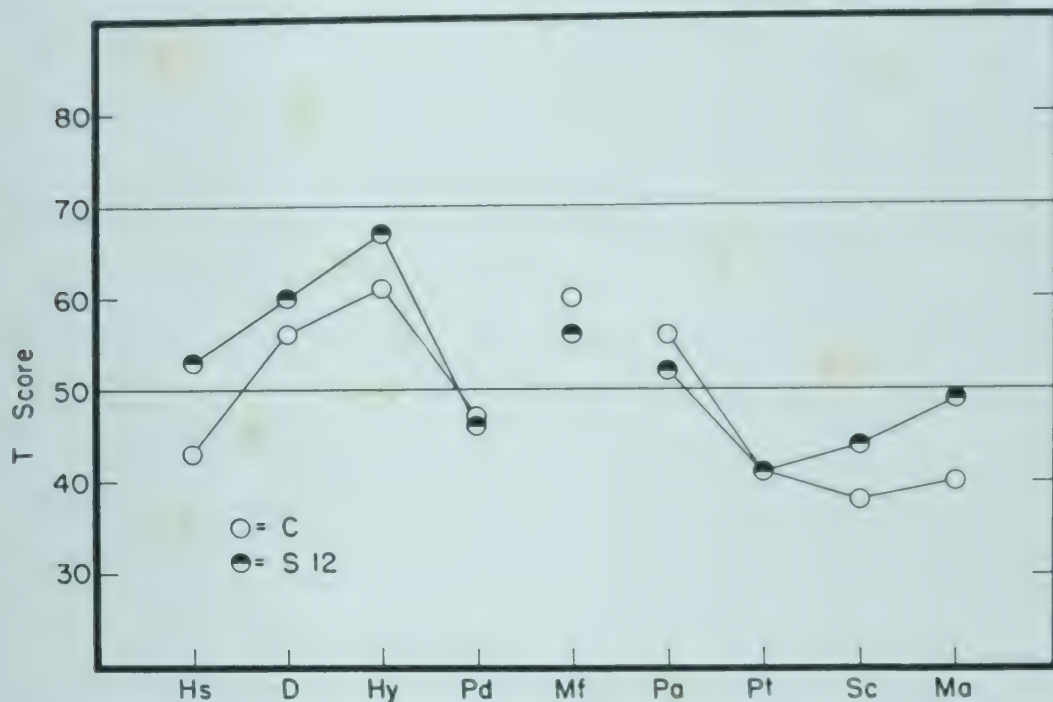


FIGURE 123. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT No. 235 during the control period (C) and after 12 weeks of semi-starvation (S12) (Minnesota Experiment).

On the neurotic end of the MMPI the subject's peak is on Hy (see Figure 123). This elevation is moderate and, taken by itself, would hardly be considered to have clinical significance. However, in view of the very high scores on one of the validating scales (the L or Lie scale), ranging from 68 to 78, the elevation in Hy assumes added significance and indicates a hysteroid temperament. In terms of the items which constitute the L scale of the MMPI, the subject appeared to himself as a "perfect" person who always tells the truth (item J42), likes everyone he knows (J47), never feels like swearing (J51), and has table manners just as good at home as when he is out in company (J54). Such an individual must be singularly resistant to self-revelation.

In summary, the restlessness, self-depreciation, and eating off diet, followed by an emotional reaction and vomiting, may be considered as evidence of a clinical neurosis. That the neurosis was mild is accounted for by the fact that the stress was terminated early, that it was mitigated by taking the extra food, and that the hematuria provided a ready-made face-saving rationalization. His ability to dissociate himself from reality (e.g. eating when in a "mental blackout") and to rationalize served as a psychological protection and allowed him to reduce his suffering by breaking the diet without severe feelings of guilt.

Subject No. 232

This man's story is presented because of his incapacity to adhere to the diet and the development of a definite personality disorder.

The 25-year-old subject was a husky athletic individual. His parents were farmers in modest circumstances. The family was large but congenial and upheld strong moral principles. The subject attended college for two years. There were many evidences of latent neurotic characteristics in his personality makeup. He was a friendly, boyish type of individual, with a strong tendency to try to appear as he thought one should. His past history is full of episodes in which he was indecisive and unable to formulate his goals clearly. His struggle with the problem of pacifism is typical. He was imbued with pacifistic principles from childhood and finally became a conscientious objector although he was never completely sure of his stand. In his autobiography he says, "With a somewhat melodramatic feeling I finally committed myself to it."

In the first few weeks of starvation the subject showed the usual reactions of hunger and loss of energy. His weight loss began to fall behind the predetermined rate starting with the fifth week (see Figure 124). By the ninth week it became apparent that he was tense and worrying considerably. After the story of No. 234 became known, the subject confessed minor irregularities of diet such as eating a crust of bread; these appeared to trouble him greatly. At this time, along with two or three others, he began to chew gum in enormous quantities

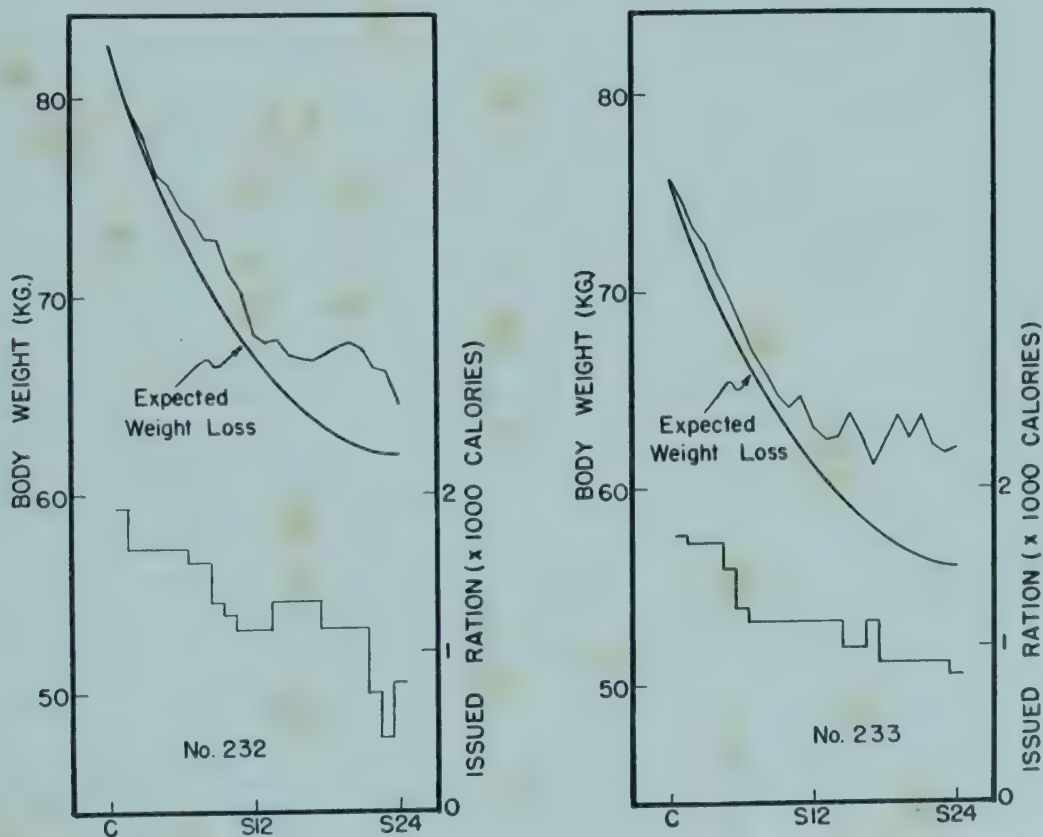


FIGURE 124. WEIGHT CHANGES AND CALORIC INTAKE FOR SUBJECT NOS. 232 AND 233 (see caption, Figure 119).

(up to 40 packages a day). By the middle of the starvation period (S12) he was in a sorry state. His finances would not stand the heavy expenditures for chewing gum, his mouth became sore, and though he made valiant efforts to control the gum chewing, it only became worse; on one occasion he was known to have stolen gum. This compulsive behavior appeared to be aimed at the alleviation of hunger sensations, at allaying nervousness, and at reducing the temptation to eat off the diet, but little beneficial effect was obtained.

During the last 6 weeks of the starvation period the man's restlessness, sense of guilt, and general nervousness increased decidedly. In spite of having very little money he paid \$10 for an old suit that was evidently of little use to him. Later he wailed, "Nobody in his right mind would do a thing like that." In interviews it was noted that his characteristic indecisiveness had become markedly aggravated. He was disgusted because of his inability to control his gum chewing. He made many ambiguous references to his previous minor dietary irregularities and talked a great deal about how awful it would be to break the diet. However, he was unable to face the issue squarely and did not make a clean confession. Instead he talked a great deal about a compulsive attraction to refuse and a strong, almost impelling, desire to root in garbage cans. Information obtained in post-experimental interviews revealed that some of his behavior was self-punishing: he had not bought good food but actually ate garbage, a sandwich he found on the ground, and a student's lunch which he had stolen.

Since his weight failed to go down to the desired level in spite of drastic cuts in his diet, he was dropped from the experiment at the end of the starvation period. This meant that he left the Laboratory and was completely relieved of all restrictions. However, his neurotic manifestations continued in full force and even increased for a while. He repeatedly went through the cycle of eating tremendous quantities of food, becoming sick, and then starting all over again. He sought interviews, complaining that he needed psychological rehabilitation. He was emotionally disturbed enough to seek admission voluntarily to the psychiatric ward of the University Hospitals. He left again within 24 hours, giving as his reason that he had to find a job. After a few days he returned, asking for further psychiatric help. During the subsequent interviews he was self-deprecating and felt confused and defeated. On one occasion he cried freely and became so agitated that he kicked over a table and broke his glasses. With characteristic inconsistency he attempted to relieve his conscience by making partial revelations but still avoided a painful complete confession. He appeared to derive little benefit from the psychiatric contacts, since he was still unable to make plans or decisions, attempting to cling to impossible solutions and face-saving devices. It took him several weeks to liquidate a few items of personal business and depart for home. Follow-up information indicates that his problem gradually subsided over a period of weeks and that he eventually made a satisfactory adjustment.

The MMPI profile for this subject (see Figure 125) indicates a definite neurotic pattern at the twelfth week of starvation, which was accentuated still further toward the end of the semi-starvation period. In addition to the elevation on the "neurotic" end of the profile, there was also a significant rise in the scores

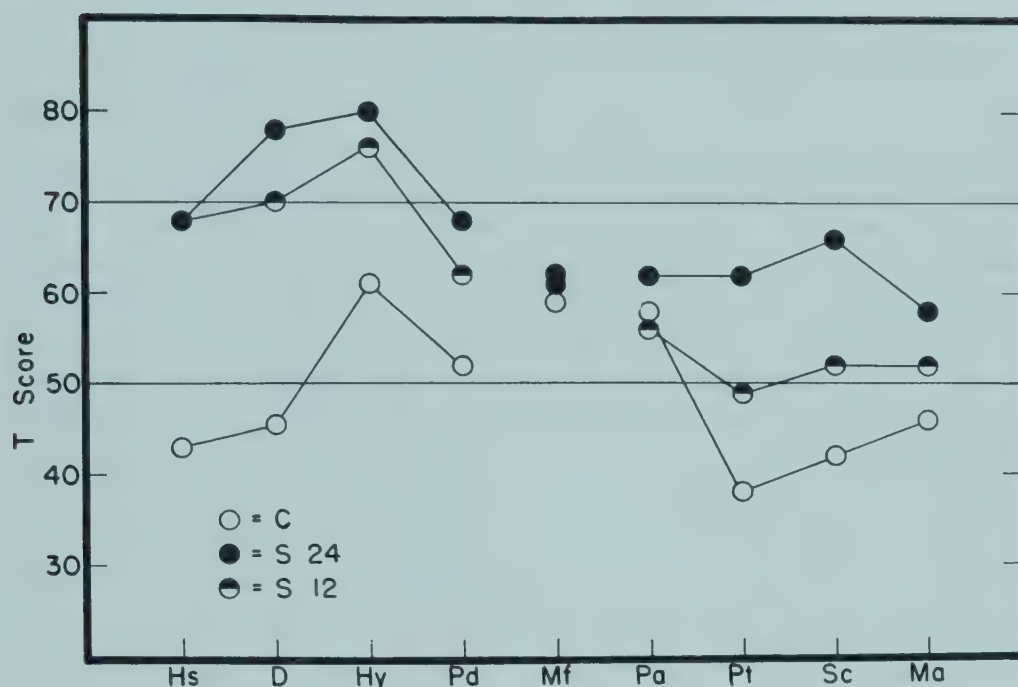


FIGURE 125. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT NO. 232 during the control period (C) and after 12 and 24 weeks of semi-starvation (S12 and S24) (Minnesota Experiment).

on the “psychotic” scales, even though without control values the semi-starvation scores would not be considered as definitely abnormal.

In summary, this subject’s latent personality weaknesses were amplified and brought to the surface by the stress. He did not have the strength to carry out the program or the capacity to decide unequivocally to get out of the unpleasant situation. Thus he developed an experimentally induced neurosis characterized by such symptoms as indecisiveness, self-depreciation, feelings of guilt, restlessness, nervous tension, compulsive gum chewing, and eating off diet.

Subject No. 233

This man’s record is somewhat similar to that of No. 232. The central feature is the probable breaking of the diet.

The two subjects were close friends. Both were athletic and less intellectual and less cultured than the average member of the group. This man was also addicted to excessive gum chewing, and he failed to lose weight in spite of drastic reductions in his diet (Figure 124). Because of this his data were not used in the final group analysis, although the subject remained in the experiment and was used for independent short-term biochemical observations.

There was only indirect but convincing evidence that his failure to lose weight was due to dietary violations. He denied eating any unauthorized food although he admitted that he was unable to control his gum chewing because of his nervousness. He appeared very depressed but was inarticulate in both the interviews and his diary, in which he generally wrote much less than the average subject. Despite the fact that there was no surface evidence of maladjust-

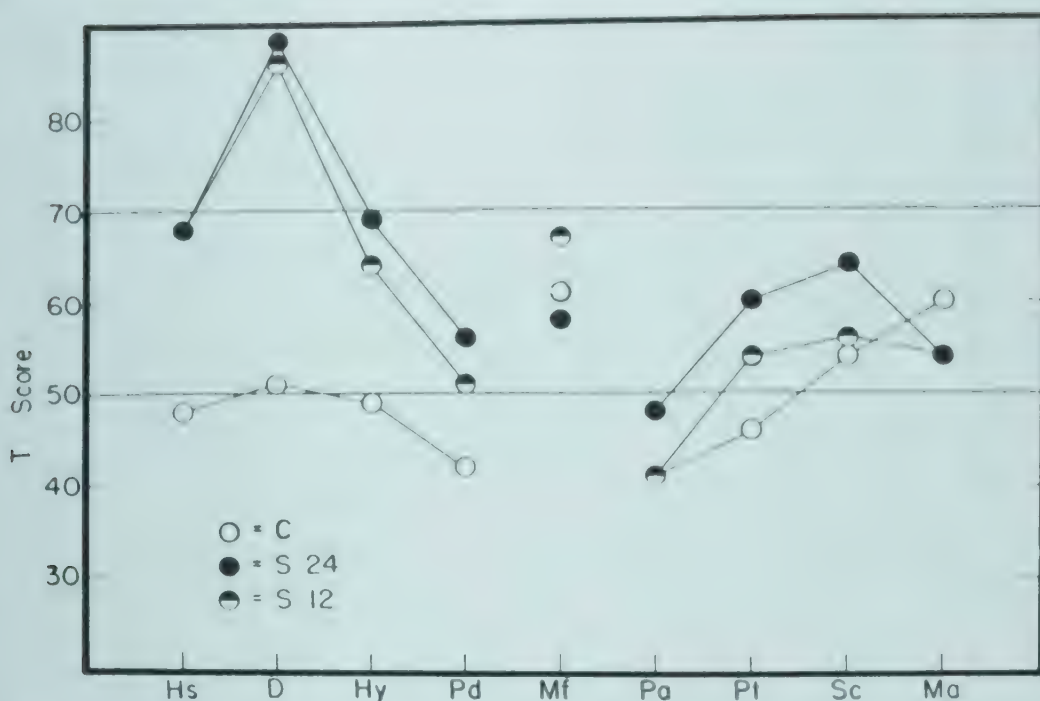


FIGURE 126. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT No. 233 during the control period (C) and after 12 and 24 weeks of semi-starvation (S12 and S24) (Minnesota Experiment).

ment on entering the experiment, his background shows ample evidence of difficulty. He had feelings of inferiority and lacked self-confidence; he described himself as disorganized, lacking in planning, and a procrastinator; he wet the bed until the age of 15 and was shy and poorly socialized until much later.

His MMPI showed a striking rise early in the semi-starvation period (see Figure 126); the control profile was normal. In this case the changes in the MMPI profile present a more correct picture of the semi-starvation neurosis than would have been obtained on the basis of diaries and interviews, in which the subject was hopelessly unable to express himself.

Subject No. 20

This subject suffered a pronounced personality deterioration culminating in two attempts at self-mutilation. The complex psychogenesis of these "accidents" justifies the presentation of the story in considerable detail.

The subject was 28 years of age; he came from a wealthy urban home. His father died when the subject was 14 years of age, but he left a considerable fortune. Although this man is the youngest of the three children, he early assumed the leadership in family matters. His mother exerted a dominating influence over her children. Her second marriage turned out unhappily, ending in divorce, but only after the stepfather had wasted the family fortune. The brother, aged 31, had been a long-standing problem, failing in academic, occupational, and marital ventures. The sister, aged 30, had remained at home in order to be a companion to her mother. The subject was quite concerned about her and feared that she was wasting her life. It is important to note that the subject's family

matters remained complicated, unsettled, and distressing to him during the time that he was participating in the experiment.

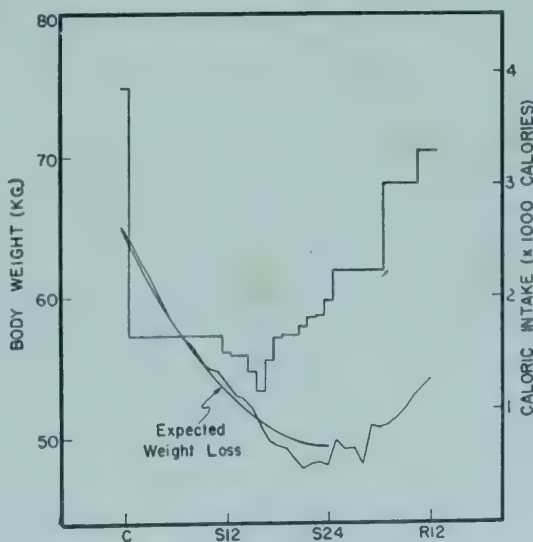
He graduated from college at the age of 23. He was an able student, especially proficient in languages, which he taught for a period of time prior to induction. However, his vocational plans were confused. He was highly intelligent, engaging, extroverted, and had a capacity for becoming well liked. He was conscientious, determined, and a hard worker, with notable humanitarian and social welfare interests, and he would be a leader in most groups. In spite of these assets he showed a peculiar immaturity for his age and background — an immaturity expressed in exaggerated standards for himself, vocational indecision, underdeveloped sex life, dependence on his family, and restlessness. Frequently he drove himself hard to complete a task, and then felt compelled to go to bed at home for an extended period of recuperation. His mother also reacts in this manner whenever she is forced to confront a difficult situation.

During the first 2 months the subject was losing weight at the predetermined rate. By the mid-point of the semi-starvation period he had definite signs of edema. Diuresis at about the fifteenth week resulted in a rapid loss of weight (see Figure 127).

His semi-starvation neurosis reached dramatic proportions. He suffered considerably from hunger, weakness, irritability, and moroseness. His eating habits became annoying to others because he spent hours in making "God-awful" concoctions and otherwise dawdling over his food. Since he had appeared at the start as *the* strong man who could "take it," the personality changes were especially distressing to him. He was hurt by his drop in popularity. However, he continued to take a strong stand until the fourteenth week, when doubts began to creep in.

"My philosophy of pushing mentally and physically is quite obviously breaking down." He felt that he had had about all he could take. Only the approaching end of semi-starvation some 10 weeks away enabled him to hold together. But his strength of character was being shaken by the temptation to escape from the stressful situation. He wrote, "I have had a horrible thought today. My cold has settled into an annoying cough, and I got to thinking how pleasant it would be to contract tuberculosis, merely for the rest and food involved in its treatment. I am terribly ashamed of the thought, but it came so here it is on paper. This awful week has undoubtedly contributed to its development. I hope more awful weeks don't produce more ghastly ideas."

FIGURE 127. WEIGHT CHANGES AND CALORIC INTAKE FOR SUBJECT NO. 20 (see caption, Figure 119).



For a time his determination rallied. He wrote that even if he developed tuberculosis he would insist on remaining in the experiment for the benefit of medical science. Shortly thereafter he attempted to burn his bridges by committing himself to an extra 2-month period of experimentation after the 12 weeks of controlled refeeding.

On the last day of semi-starvation he collapsed on the treadmill. Although this should not have been considered unusual in view of his physical weakness, he suffered an acute emotional upset because he felt he had failed to live up to the standards he had set for himself. "I've been miserable all day because I fell down on the job. To have held out this long and then on the last day to fold is more than discouraging. I feel as though I had failed in someone's trust. I started bawling like a baby and kept it up for a long, long time. I have never been so ashamed of any performance as I was then, but had completely let go of myself and couldn't do anything but go on sobbing . . . ever since I've felt like a quitter and I hate quitters. That wonderful last day has been spoiled. I only hope a return of strength will help me look a little less tragically on today's flop."

While the subject was making such a struggle to appear strong during the semi-starvation stress, he had failed to anticipate that the 12-week rehabilitation period might be little better than the preceding starvation. It was his bad luck that he was assigned to the next to the lowest caloric group. The going was tough for him, and the idea of continued participation in the experiment was becoming unbearable.

At the end of the first week of rehabilitation he injured his left hand when his automobile slipped off the jack. One finger was torn three fourths off at the distal phalanx and required outpatient surgical care. He made it appear that this was an accident but confided the truth to one Staff member. In order to get out of the experiment he had attempted to mutilate himself; he had done an incomplete job because he lost his nerve at the last minute. The injury was not serious enough to warrant his release from the experiment. His psychologic tension was not relieved after the accident or following his confession.

The next week his diary indicated that he was painfully aware of the fact that he could no longer hold up during this unexpected continuation of the stress, let alone appear as a *strong* man. "This has been my worst week of the entire experiment. It has been caused probably by the realization that I have been subconsciously setting my sights at the end of starvation, considering getting through that stress as the job before me and expecting a letdown and rest after it. Of course such a letdown was not possible; in fact, the most important part of the experiment is the present one, and the job won't be completed until October (10 weeks away). Unfortunately, I wasn't ready for the strain of rehabilitation and when it came I almost cracked under it. I have had doubts recently about my physical and mental ability to continue the experiment. Since quitting is so obviously contrary to my basic desires and common sense, I have run into an awful conflict that has been intense enough to make me wonder whether I am losing my mind. I can't promise that I won't blow up in some manner, letting off the steam that is still within me."

Two days later he wrote, "God grant us no more weeks like this one. Physically I have felt much as at the end of the starvation period. Mentally I have

been more depressed than ever in my life. I have decided to will some cheerfulness and pep into myself. We shall see how that makes out. Unless I force myself to be cheerful, I feel sure that I'll sink into a hopeless slough of despondency which could lead into a complete mental and physical breakdown. I am scared of such a thing and am determined to avoid it if possible. I think the only way to avoid it is to make myself do things I don't feel like doing, keeping busy at something, anything at all, so long as I am busy. Inactivity will drive me nuts."

While he was in this unhappy state of mind, his sister came to visit him. Their visit started off badly because there were many troublesome family affairs to be discussed. The next day, in spite of this additional stress, his spirits lifted remarkably. That evening he and his sister went to the home of a friend. While his sister had dinner with their hosts, the subject went into the yard to chop wood as he had often done before. He somehow managed to chop off three fingers of his left hand. He was given emergency surgery in the Students' Health Service at the University Hospitals, where he remained for five days.

On the day following the accident the subject, while mildly distraught, talked freely and showed a partial insight into the psychodynamics involved. The following is a verbatim record of an interview:

"I've always thrown myself into everything I did and have done it very hard. Afterward I have reacted with fever and collapse and have been babied by my mother. I have recently had the stress of this pneumonia and then the last month of semi-starvation was very tough, especially the edema. On top of that I've had a difficult home situation. I owe it to my mother and sister to spend time at home. I asked to be taken off the list of those going abroad since my mother's affairs are more important. Then rehabilitation started. I had looked to six months of starvation as a job to be done and I did it. But then I had no chance to relax and rest and let down. When rehabilitation started, I was still hungry. It was really more starvation. In fact, I suffered from more hunger because I could not take food out [to make "sandwiches"] as I had before. I was blue over the whole thing. I was in a weird frame of mind. I thought that there was only one thing that would pull me out of the doldrums, that is release from C.P.S. I decided to get rid of some fingers. Ten days ago I jacked up my car and let the car fall on these fingers. It missed them all except it crushed the end of one finger [same hand]. That's not normal. It was premeditated.

"Since then I have begun to worry about my state of mind. I have also worried about the family; my brother's wacky, my sister's very worried over my mother and maybe there's something wrong with me too. I've always been able to sleep, but not last week; I tossed and turned. I tried to do some reading but all I could do was think of home. I had so little control over my mind that I was afraid I would lose it. One morning at breakfast I came closer to an act of violence than I ever had before. Someone across the table — I can't remember who — will never know how close he came to having a tray smash down on his head. He'd done nothing. I just wanted to be at my mother's and not at the breakfast table in Shevlin Hall. It was all going on in my mind. I just felt that I had cracked. Finally, in bed at night I tried to be as objective as I could and I've managed to force myself to stick until July 29 [end of semi-starvation]. On the last day of semi-starvation, well, I didn't finish it . . . I only lasted 10 seconds

on the treadmill and that upset me and I bawled for a half hour and then more. I was so disappointed to flop at the last test. As for this [referring to the loss of the fingers], I don't know. I had made up my mind to stick it through to October 20. I felt I'd be demobilized by the end of my leave. I felt there'd be no point in this at this time when the war is over. How silly it had been to drop a car on my fingers, I thought. I certainly had no idea I'd do this [*sic!*] I am now back at the place I was; that is, I'd like to get a 4-F and go home. I'm afraid. What will I do next?"

Although the subject was kept on the same caloric level in the hospital, he did receive a few variations in diet items (e.g. fruit, etc.). He appeared to enjoy the experience immensely. The bed rest, attention, and freedom from routine during a few days of hospitalization seem to have satisfied his immature, dependent needs and to have served as a satisfactory substitute for home and his mother.

After release from the hospital, the subject was persuaded to remain in the experiment and was able to carry on during the remaining two months. The pain he suffered may have served as punishment for his failure to perform up to expected standards and for his desires to leave the experiment; it may have expiated his sense of guilt. Also, he was being slowly rehabilitated physically and was approaching the end of the regimentation imposed by the experiment and the CPS assignment.

In subsequent interviews he almost completely repressed the purposeful nature of the "accident." Although he could not clearly explain or describe exactly how it happened, he gave "rational" suggestions: he was too weak, he had poor control, it was uncomfortable to hold the ax with both hands because of the sore finger on the left hand (previously smashed in the auto-jack incident), the ax must have hit a branch, etc. He argued strongly that the accident had no personal motivation, yet he did make this comment: "I wasn't myself for two weeks. I may be more valuable to the experiment than if I hadn't done it." From this time on he carefully avoided all mention of the accident, though he continued to talk and write freely on everything else. He was puzzled over a newly acquired aversion to psychology and psychologists; this appears to have been a defense against self-revelation.

It is of psychological interest that the pattern of self-mutilation appears to have been suggested by an experience two years earlier which he never once mentioned while at the Laboratory. At a previous Civilian Public Service assignment he gave first aid to one of his close friends who had lost several fingers in a buzz saw; this friend was subsequently given a 4-F and released from CPS.

The MMPI profile of subject No. 20 taken during standardization is normal, while the one taken at the end of semi-starvation is severely neurotic in type (see Figure 12S). At the second week of rehabilitation the D score had further increased by nearly one standard deviation and, more significantly, the psychotic end of the profile showed a marked rise. Such a profile indicates that the subject was under a severe stress which he was not able to handle. This profile was obtained four days after his first accident and six days before his second one.

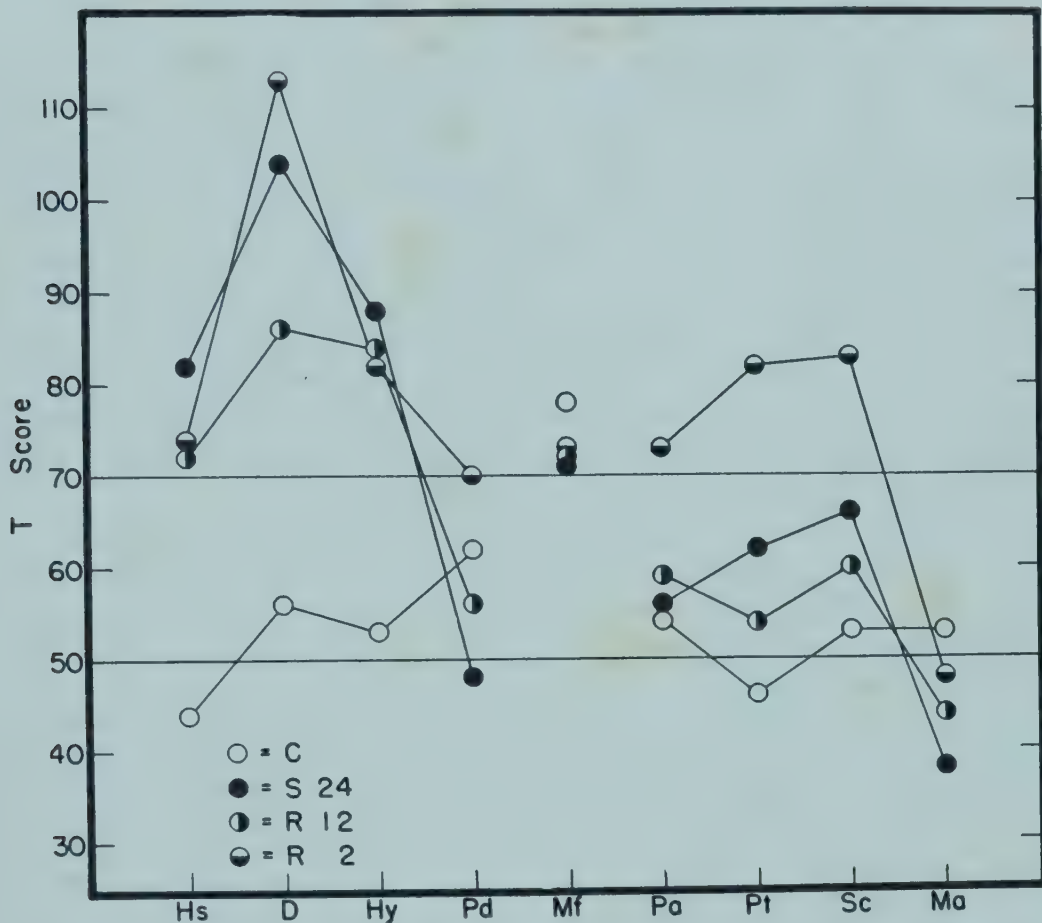


FIGURE 128. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT No. 20 during the control period (C), at the end of semi-starvation (S24), in the second week of rehabilitation (R2), and at the end of controlled rehabilitation (R12) (Minnesota Experiment).

The scores at the twelfth week of rehabilitation indicate a slow return toward normality.

In summary, subject No. 20 displayed in a severe degree the usual personality changes of "semi-starvation neurosis." The physical deterioration conflicted sharply with his pattern of always being the strong man. By expecting rapid rehabilitation, he misgauged the duration of the stress; because of his "all or none" pattern, he was particularly unprepared for this and was unable to make the necessary psychological adjustment. In addition he suffered severely from a conflict between the desire to escape from the painful situation and the desire to save face. The psychological situation was further complicated by distressing home conditions. All this led to a severe emotional conflict.

The first attempt to solve this conflict was through deliberate self-mutilation (smashing his hand). After this abortive attempt he was disgusted with himself, miserable and depressed. The second "accident" appears to have been brought about by more unconscious mechanisms. The action, while not providing a solution for the underlying problems, did have a therapeutic effect which enabled the subject to complete the experiment.

Accidents and Undernutrition

It may be useful to consider at this point the general question of accidents and undernutrition. In the Netherlands industrial accidents rose from 22.9 per thousand men during the first 5 months of 1939 to 138.5 during the corresponding period in 1943. The nutritional deterioration at that time was inconspicuous, but Penris (cited by Dols and van Arcken, 1946) concluded that the nutritional state was mainly responsible for this increase; the emotional stresses of war and of the German occupation must also be taken into account. More valid evidence that undernutrition makes people prone to have accidents at work was brought out by Leyton (1946), who attributed the accident-proneness to the slowness of movements and the prolonged reaction time. He observed also that the shuffling gait of starving men caused them to stumble over small obstacles and made it difficult to walk on rough ground without mishap.

Curtin (1946) points to fatigue and weakness as factors contributing to the accident-proneness of the malnourished men held in Japanese camps for Allied prisoners of war. Some of the accidents resulted from faulty machinery and lack of goggles during work in which arc-welding torches were used. On the other hand, purposive accidents also occurred; the prisoners would intentionally injure themselves in order to be released, at least temporarily, from work. Nutritionally they did not gain since the food received by sick prisoners was usually less, sometimes only half the ration received by men who were able to work. These "accidents" were intended to result in minor injuries but sometimes the trick proved only too successful. Curtin reports that one prisoner had his right hand and forearm torn and crushed by machinery in a brick factory and that another suffered a traumatic amputation of two toes and part of his foot.

It is evident that besides the physical (physiological) bases for increased accidents among starving people, purely psychological factors must be considered. The general state of apathy is associated with inattention to the details of the surrounding world, including potential dangers of accident. Also, it is difficult to draw the line between pure accident and deliberate self-mutilation. The belief or hope that perhaps a physical injury may bring an improvement in the nutritional situation is not difficult to entertain, whether or not such a result is really probable.

Subject No. 5

This subject developed neurological symptoms, probably hysterical in origin, in addition to the usual semi-starvation changes.

This 29-year-old man came from a rugged background. His parents were mill hands who lived in poverty most of their lives. The subject was raised in the slums and was often placed in boarding homes while his mother worked. There was no religion in the family. The father showed no interest in the home and the brother had many traits of a psychopath. The subject was ashamed of his father and had no close feelings for his brother.

As the patient matured, he appears to have made a fairly good adjustment to life, emancipating himself from home influence. After some deliberation and conflict he chose the ministry as his vocational goal. In temperament he appeared extroverted. He was highly intelligent and had ability as an actor. On the

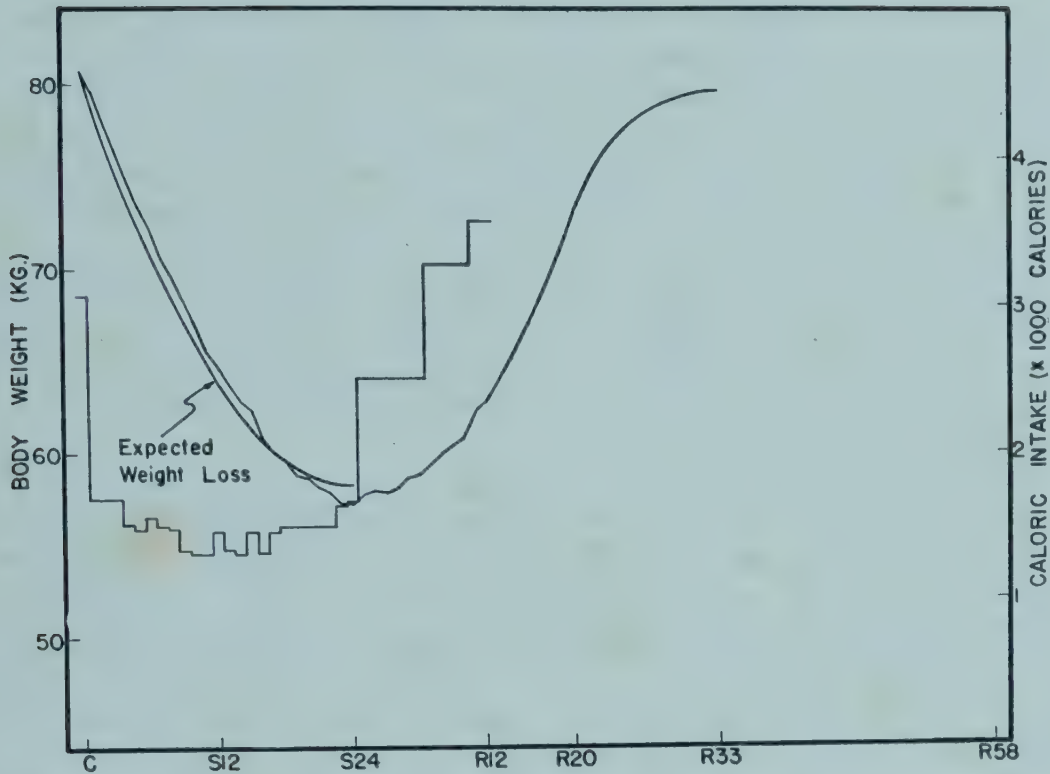


FIGURE 129. WEIGHT CHANGES AND CALORIC INTAKE FOR SUBJECT No. 5
(see caption, Figure 119).

surface he appeared easygoing and good-natured, but he was restless and often became tense. Most of his previous Civilian Public Service experience was in mental hospitals, where he had served as an attendant. This contact stimulated his interest in psychiatric disturbances and led him to an unceasing psychological analysis of himself and of others. He was a person of strong feelings and developed strong likes and dislikes. Although he tried to be objective, his personal antipathy toward the interviewer to whom he was first assigned was so intense that a change was necessary.

The subject was overweight and was required to lose weight during standardization (from 83.6 kg. to 80.8 kg.). Since he was still above normal weight for his age and height, his weight loss was placed at 29 per cent as compared with 24 per cent for the group average (see Figure 129). During the first half of semi-starvation he developed the physical symptoms common to the group, but he suffered less psychologically. He was so elated over the happy progress of a new and promising love affair that he thought and talked of little else.

Midway through semi-starvation the girl broke their engagement. Not only was this in itself a severe blow, but he now became fully aware for the first time of the disquieting effects of the starvation stress. His dreams about food became more intense. His depression and irritability were marked. He was impatient and strongly resented the restrictions incidental to the experiment.

During the next four weeks he continued to be in a turmoil over his unhappy romance. He met his ex-fiancée on several occasions in an attempt to straighten out their affairs. These experiences were not very satisfactory, and he continued

to feel bitter over "what might have been." Although he decided to "wash things up, clean, neat and final," he continued to see her at every opportunity.

Since he considered himself a sophisticated and worldly individual, he was dismayed to find that he suffered emotional tension and peculiar feelings every time he met the girl. They saw each other often since they both participated in the activities of the University Theatre, where they had met originally. He was particularly alarmed by the fact that on several occasions he found himself on the verge of tears. These emotional experiences were nearly overpowering, came unexpectedly, and always occurred in the theater or other situations associated with the romance. As time went on, the subject became better able to control his outward behavior and feelings, and he found some solace in the company of his ex-fiancée's sister.

In the eighteenth week he reported in his diary that his spirits were much improved but that he was beginning to experience some difficulty in walking: "My right foot seems unhinged at the ankle. When I step on my heel, the toe comes down with a slap as if I had no control of the muscles." At the same time he noted a transient numbness of the right thumb. Two days later he noted for the first time that he was able to converse with his ex-fiancée without emotional distress. "No feeling aroused at all. She might just as well have been any one of a dozen other girls I know fairly well."

This same day he reported to the Staff his new physical complaints. The symptoms included "numbness" near the base of the index finger of the right hand and hypesthesia on the anterior aspects of both legs, extending from below the knee to the toes. He also complained of a peculiar weakness of the right ankle, which "gave way" unexpectedly. All these symptoms recurred intermittently.

Neurologically, the subject's condition was objectively normal on all three occasions on which he was examined. He was able to distinguish sharp from dull and to recognize light touch at all times. The hypesthetic area on the hand and the sensory disturbance of the left leg disappeared permanently in a few days, and the other symptoms gradually faded out over a period of ten weeks. The record of the routine neurological re-examination made during the fourth week of rehabilitation indicates that though the hypesthetic area on the right leg was still reported as present, the subject stated that it no longer attracted his attention. At no time did any of these symptoms cause real discomfort to the subject, and he did not show concern over them. He reported their presence in a matter-of-fact way as a part of the experimental routine.

That the neurological symptoms experienced by this subject were hysterical in nature is supported by the negative objective neurological findings and the vague outline, indefinite localization, and intermittent character of the sensory disturbances. They were preceded by a period of relative emotional calm in reference to his love affair. The subject, who had studied abnormal psychology in college, considered the condition as probably hysterical although he was puzzled as to how one so sophisticated as himself could have hysteria.

His MMPI was normal during control (see Figure 130). The semi-starvation profile, obtained after he had had symptoms about five weeks, showed the typical elevation on the neurotic end of the curve with a peak of 78 on Hy.

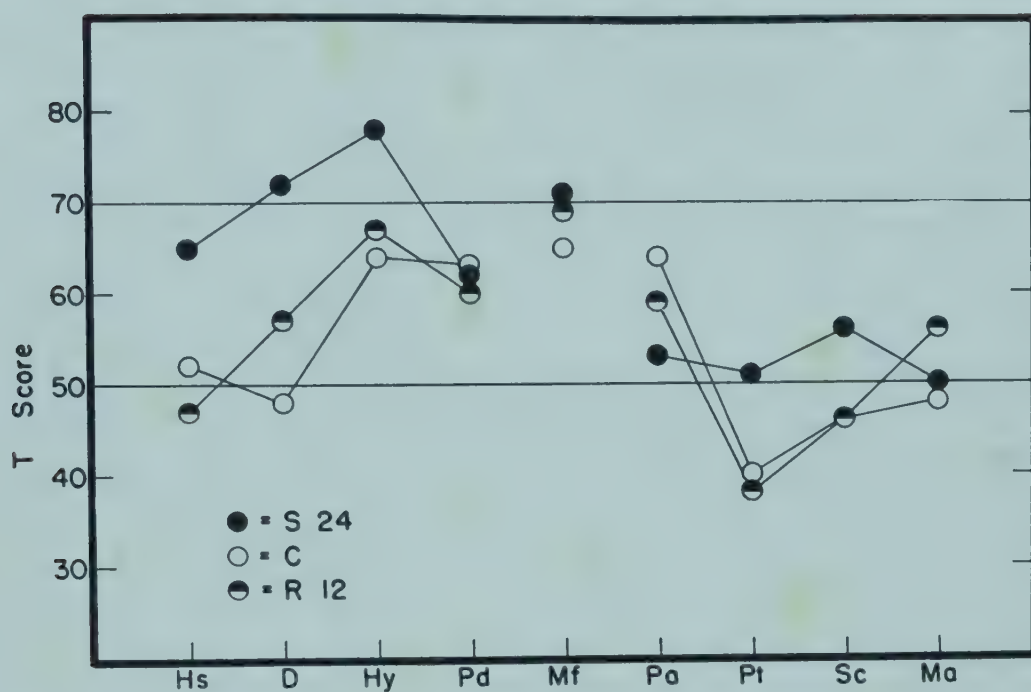


FIGURE 130. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT No. 5 during the control period (C), after 24 weeks of semi-starvation (S24), and at the end of controlled rehabilitation (R12) (Minnesota Experiment).

Although it seems unlikely, it can be argued that there may have been neurologic, nutritional, or other physiological factors responsible for the development of the sensory complaints. Even if this were so, it is our opinion that these became the focus for the hysterical conversion that played an important role in the production of the final clinical picture.

Subject No. 130

This subject had a history of personality difficulties when he entered the experiment; these became aggravated during the stress. The final result, however, appears to have been a net gain.

This 24-year-old pre-theological student was born and partly raised in India, where his parents were missionaries. Part of his childhood and adolescence was spent in the United States. His background history is filled with evidence of adjustment difficulties which led him to change schools many times. In childhood the subject suffered from terrible nightmares: "To this day [they] send shivers down my back just to think of them." He struggled over autoeroticism and feared that he had stronger sex desires than most people. While in college he was told by the school psychiatrist that he was "a mess." Yet in spite of marked neurotic qualities he had a good record of accomplishment.

He described himself as having few close friends, being shy and submissive, and having a marked inferiority complex. "I am conceited, self-centered, inconsiderate, tactless, and blunt. I have a lousy personality even though I know how to get along with most people and can hold friends." In recent years he had had several periods of mild depression alternating with periods of mild elation. Just

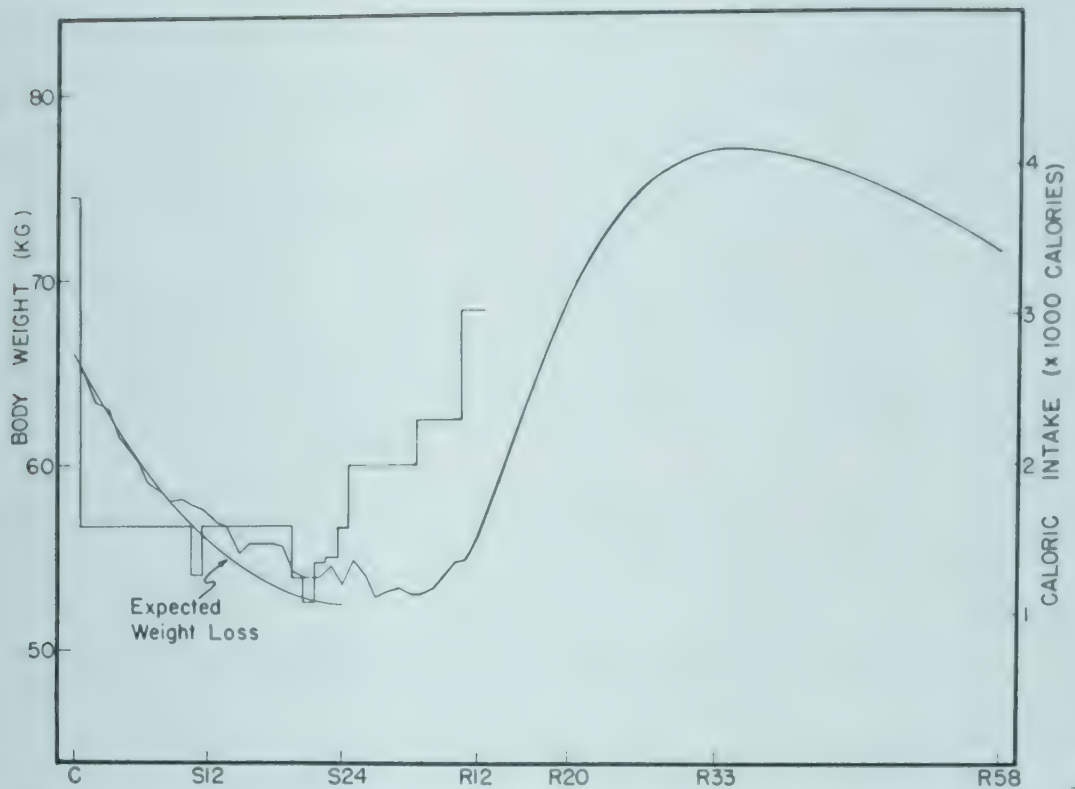


FIGURE 131. WEIGHT CHANGES AND CALORIC INTAKE FOR SUBJECT NO. 130 (see caption, Figure 119).

prior to his admission to the experiment he worked as an orderly in a mental hospital. At first he was in an upswing; he enjoyed his work and founded and edited an institutional publication. Some of the articles he wrote contained such "scorching invective" that he was nearly dismissed from his job. The elated phase was soon followed by a depression that lasted several months and was more severe than the preceding ones.

The history of this cyclothymic disorder was taken into account in considering the subject for the experiment. It was finally decided to include him because of his assets. He had insight into his problem, gave evidence of strength of character, had a good record of accomplishment in spite of his personality handicaps, and gave the clinical impression that he would be able to complete what he started.

Objectively, the patient did very well during the entire experiment. He had the usual physical symptoms and slightly more than his share of irritability and hostility, the latter being expressed especially toward one of the generally unpopular men (No. 29). He developed severe edema, reflected in his weight curve, which showed a loss of weight on refeeding (see Figure 131).

The subject experienced a number of periods in which his spirits were definitely high; he associated this with discovering that he could take the stress of the experiment better than many of the men toward whom he had previously felt inferior. These elated periods alternated with times in which he suffered "a deep, dark depression." None of these mood swings, either up or down, lasted more than a few days.

Toward the end of the starvation stress the subject felt that he had reached the end of his rope. He expressed the fear that he was going "crazy." (No. 20 was the only other subject to express this fear.) He felt that he was losing his inhibitions. He looked pained and depressed much of the time. On many occasions during the last 3 or 4 weeks of semi-starvation he had impelling desires to smash or break things. At no time, however, were these impulses carried into action. In rehabilitation he was in the lowest caloric group, but he carried on in spite of the resulting slow rate of recovery. As time went on, his mood swings lessened in intensity and frequency.

After the experiment was all over, the subject felt he had been personally strengthened by the ordeal. "It undressed us. Those who we had thought would be strong were weak; those who we surely thought would take a beating held up best." The personal satisfaction he got from the suffering and the successful completion of the experiment is clearly indicated in his diary: "I am proud of what I did. My protruding ribs were my battle scars. My abnormal conduct in society also was where I was hit. I am proud of these. I am glad I acted like a fool, that I became so weak I could hardly turn over in bed, that I thought with my stomach instead of my head. It was something great, something incomprehensible."

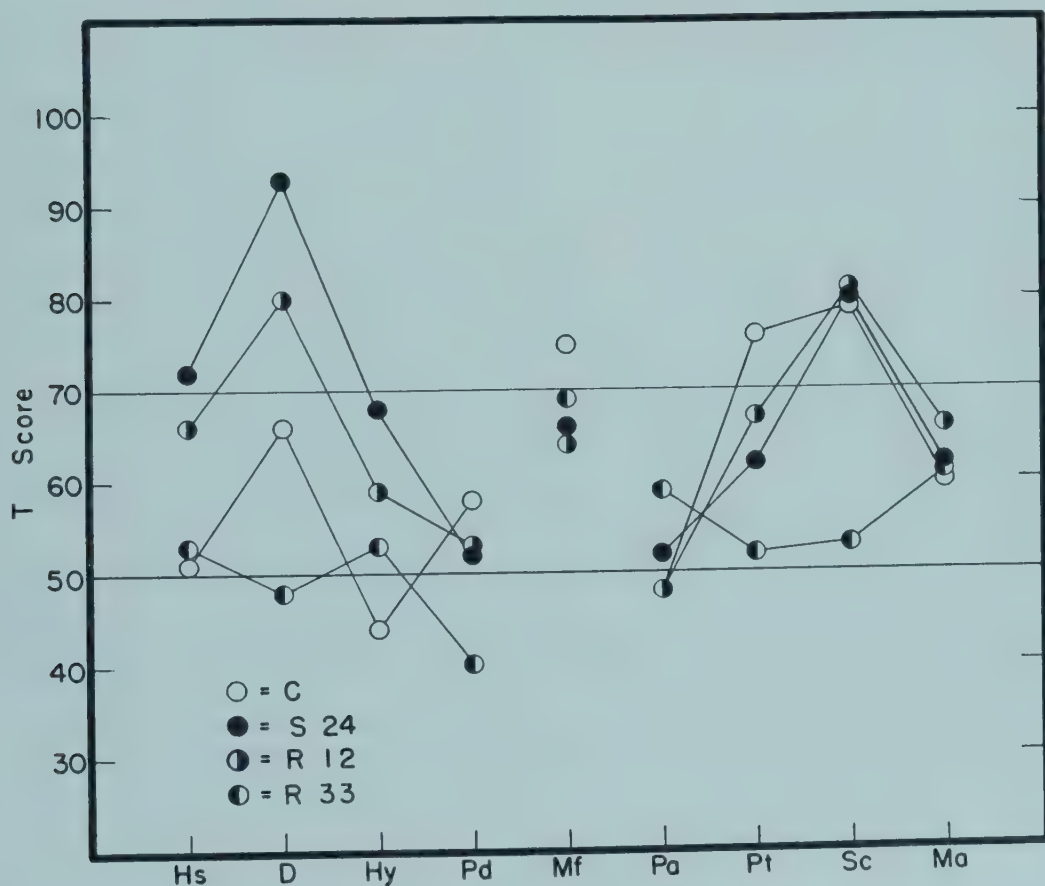


FIGURE 132. SCORES ON THE MINNESOTA MULTIPHASIC PERSONALITY INVENTORY FOR SUBJECT NO. 130 during the control period (C), at the end of semi-starvation (S24), and after 12 and 33 weeks of rehabilitation (R12 and R33) (Minnesota Experiment).

Four MMPI profiles are presented for this man (see Figure 132). The pre-starvation profile shows an elevation on the "psychotic" end. This was consistent with his cyclothymic pattern. At the end of semi-starvation the profile shows an added neurotic response of considerable severity. In other words, the whole personality is involved. By the twelfth week of controlled refeeding there was a noticeable improvement in those symptoms which were brought out by the semi-starvation stress. By the thirty-third week of rehabilitation (that is, after 21 weeks of complete freedom) his scores on the psychoneurotic scales of the MMPI are as good or better than his pre-starvation values. In addition, there is a striking improvement in the scores on the psychotic scales of the inventory. This suggests that the experience of going through the experiment had significant therapeutic value for the subject, although the permanency of this improvement cannot be predicted on the basis of the information available.

Comment

In addition to the common ("normal") psychological reactions to the semi-starvation regimen, the subjects whose histories are presented in this chapter developed unusual or severe neuropsychiatric disturbances. A response was considered "abnormal" if it appeared to be inefficient, rare, exaggerated, or contrary to the main purpose of the experiment.

Excessive gum chewing (subjects 232 and 233) was one of the reactions that may be considered as an "inefficient" mechanism. It was started in an attempt to alleviate hunger and nervous tension, but it was continued compulsively in spite of the fact that it failed to give the desired result. This symptom occurred in great intensity in four subjects; two of these failed to adhere to the semi-starvation diet.

The subject who chopped off his fingers certainly showed a reaction that was both unusual and inefficient since it was a roundabout way to achieve the end in mind. The hysterical sensory disturbances in subject No. 5 can also be classified as unusual.

Eating off the diet was contrary to the purpose of the experiment. A strict adherence to the dietary regimen was an essential criterion of conformity to the social values of the subject group, and those who did not adhere to the agreed restrictions exhibited a behavior pattern similar to ordinary antisocial activity. It should be emphasized that with one or two exceptions this did not consist of a rational act of procuring ordinary food such as one might obtain in a restaurant. On the contrary, the subjects' inefficient and neurotic approach to the solution of the problem of hunger is shown by the fact that those who broke diet ate garbage, raw rutabagas, infinitesimal amounts of food, or in other ways attempted to minimize their behavior discrepancies and expiate their guilt.

Psychological Effects—Interpretation and Synthesis

STARVATION affects the whole organism and its results may be described in the anatomical, biochemical, physiological, and psychological frames of reference. In scientific reports attention has been concentrated generally on the purely physical aspects. On the other hand, the accounts by non-technical observers emphasize the complaints, sensations, and behavior, as well as the superficial appearance, of famine victims. The psychological changes induced by undernutrition, though more difficult to measure, seem to be just as typical as are the physical changes. Field reports are unanimous in registering a set of complaints that characteristically appear early in uncomplicated semi-starvation: feelings of weakness, hunger pains, dizziness and blackouts upon standing up suddenly, and increased frequency of urination (cf. e.g. Simpson, 1946). Additional symptoms, including profound changes in behavior and personality, are noted if the semi-starvation is prolonged.

Starvation is a fundamental biological stress and injury. At the same time, except in a few controlled experiments, it is associated with the social (and hygienic) consequences of a major catastrophe, so that the emotional impact is complicated. As Sorokin (1942) has pointed out at length, man's behavior and emotional responses to a catastrophe have certain common features and tendencies regardless of the character of the catastrophe. Besides these common features, starvation tends to produce a typical psychophysiological picture because the progressive anatomical and biochemical changes produce a dominating set of sensations, drives, and limitations to physical function. Hunger pangs, coldness, weakness, and lack of endurance cannot be constantly experienced without producing a set of mind and a direction of attitude. Finally, it is legitimate to speculate on the possibility of effects generated within the central nervous system itself by the physical changes which occur there; the tissue of the central nervous system is resistant but not wholly immune to morphological alteration by starvation (see Chapter 9).

In famine and other "natural" conditions of starvation there have been no systematic studies of the psychological consequences, and the accounts of complaints and behavior are far from satisfactory for a detailed analysis. The extent to which it is now possible to draw a meaningful and detailed picture of the psychology of the starving man in general—and of the markedly undernourished man when he is being rehabilitated nutritionally—depends in large part on the extent to which the findings in the Minnesota Experiment can be considered to have general application.

This question arises repeatedly in each of the different aspects of the biology of human starvation discussed in this book. The attempt to answer it here may be approached in two ways. First, the conditions of the experiment may be compared with those of natural starvation. With regard to the physical extent of starvation, as indicated by weight loss or tissue destruction, and its duration, the Minnesota Experiment was comparable to a great many, perhaps the majority, of the experiments of nature. On the other hand, the emotional situation might seem to have been quite different in that the Minnesota subjects had a basic assurance of ultimate safety and were spared the psychic trauma of violence and brutality which so frequently accompany natural starvation, at least in modern times. It can be argued, however, that the strain of starving voluntarily in a land of plenty produces an emotional conflict which in itself could act as a very powerful stress; these points will be examined in more detail later in the chapter. Obviously there is no way to compare, on a priori grounds, the total emotional impact of the Minnesota Experiment with the experience of a similar degree of weight loss, say, in the Netherlands in the winter of 1945.

The second, and scientifically sounder, approach to this question is to compare, point by point, the findings in the Minnesota Experiment with the available data in natural starvation. In previous chapters this has been done in detail with regard to anatomical, biochemical, and physiological features. The basic biological picture presented by the subjects in the Minnesota Experiment is beyond any doubt essentially comparable to that seen in naturally occurring caloric undernutrition; such differences as appear – and they are neither numerous nor apparently very important – find a ready explanation in secondary peculiarities such as filth and specific qualitative deficiencies in the diet (these latter in Asia). In spite of the paucity and imprecision of psychological data from the field, they too do not appear to deviate widely from the results in the Minnesota Experiment. The items of complaint and their relative frequency and intensity seem to be entirely similar if not identical. The Minnesota men exhibited symptoms of depression, irritability, “nervousness,” and general emotional instability. Social withdrawal, narrowing of interests, obliteration of sexual drive, and difficulty in concentration were prominent. Food and eating became their dominant concern. In conversation, speech was slow but did not show evidences of faults in memory or logic. This description could easily serve as a synthesis of the results of “natural” starvation as recorded in scores of reports. As far as the evidence is available on the same points in natural starvation and in the Minnesota Experiment, there are no significant differences. It would seem entirely justifiable, then, to believe that the Minnesota data can throw light on those questions on which field reports are silent or inadequate.

There are practically no data or even casual opinions in field reports regarding the course of recovery in the psychological characteristics. In the Minnesota Experiment psychological recovery was, if anything, somewhat faster than physical recovery, but both required many months of unlimited good diet. In the first few months the course of recovery paralleled, in general, the level of caloric intake; within the ranges tested, the special supplements of vitamins and proteins had little or no effect. The feeling of well-being, range of interests, emo-

tional stability, and sociability were regained more rapidly than strength, endurance, normal eating habits, and sexual drive.

In famine and natural starvation generally, individual behavior and personality appear to show greater than ordinary variability. This was clearly the case also in the Minnesota Experiment and in some respects could be documented by statistical analysis. The severity of the stress of the experiment removed the superficial façade, the *persona*, and brought out into sharper relief the individual strengths and weaknesses. The reaction was essentially similar to the differential responses of an apparently homogeneous population to any other stress. In war, for example, individual personality differences become more manifest under combat than during army routine. That individual differences increased under the experimental stress of semi-starvation is demonstrated by the rise in the standard deviation of the individual scores on the "neurotic" scales of the MMPI. The standard deviation of the Hypochondriasis scores for the 32 men who completed the experiment increased from 3.4 in control to 6.6 at the end of the semi-starvation period (S24). For Depression the corresponding values are 6.5 and 12.1, for Hysteria 6.1 and 8.2. Similarly, in fatigue resulting from intensive visual work under inadequate illumination it has been observed that the individual differences in performance tend to become magnified (Brožek, Simonson, and Keys, 1947).

In the Minnesota Experiment the personality changes were "psychoneurotic" in type and, although not grossly pathological, rendered the men increasingly ineffective in their daily life. Changes in the direction of psychoses were rare. Here again the similarity to what may be deduced from the field reports is evident; psychotic behavior and symptoms in famine are never common though personality changes are prominent. The famine victim *in extremis* may be delirious, confused, and even suggest amentia, but these are terminal phenomena rather commonly observed in the premortal stage of any prolonged and completely exhausting illness or injury. Perhaps the outstanding feature in both famine and the Minnesota Experiment is depression and apathy.

Apathy and Irritability in Semi-Starvation

In the Minnesota Experiment during the starvation phase of the study both an increase in irritability and a general apathy were observed. The same combination is repeatedly mentioned in the field reports. It was seen by Markowski (1945) among semi-starved prisoners of war who exhibited abnormally strong reactions, coupled with a prevailing tendency to depression. In Holland during the famine in the spring of 1945 the semi-starved individuals easily lost self-control and appeared "excitable"; on the other hand, they were sleepy (Dols and van Arcken, 1946). In advanced stages of semi-starvation apathy becomes the dominant characteristic (Debray *et al.*, 1946).

This curious combination of apathy and irritability in starvation may at first appear baffling. Pavlov and his students interpreted the phenomenon in terms of the general lowering of the functional capacity of the brain; the apathy, narrowing of interests, and somnolence point to a depression of the processes of excitation; the irritability reflects weakening of the processes of inhibition (see Rozental, 1922, p. 157). In the studies on conditioned reflexes in dogs which were

carried out in the Physiological Division of the Institute of Experimental Medicine at Petrograd during the starvation period of 1918-19, the loss of internal inhibition, required for elaboration of discriminatory responses to a pair of stimuli, was the earliest neurophysiological symptom of semi-starvation deterioration. The fact that new conditioned responses could be obtained only with difficulty or not at all was taken as indicating a lowered reactivity of the central nervous system.

This explanation admittedly has the appeal of theoretical simplicity. However, the same final picture might result from a completely different set of factors than those considered by Pavlov's students. One specific factor which affects the secretory activity, used as a criterion of the functional status of the central nervous system, is a changed intake of water. In Kleitman's experiments on dogs (Kleitman, 1927) the average daily water intake during 12 to 18 days of starvation was 148 cc. as compared with 356 cc. for the days when the dogs were fed. The study on water intake in starvation was really an afterthought. It followed the investigation (Kleitman and Crisler, 1927) in which a depression of the salivary conditioned reflex in starvation was obtained, thus seemingly confirming the work done in Petrograd. There is no information on water intake in Pavlov's dogs, and Kleitman's findings throw definite doubt on the interpretation of the data proposed by the Russian workers, even when one disregards the question of the validity of Pavlov's basic explanatory concept of excitation and inhibition. In observations on man during semi-starvation, an increase, not decrease, of water intake is the general rule.

"Semi-Starvation Neurosis"

In the Minnesota Experiment the personality alterations were induced by the starvation regimen and reversed by the diet therapy, and can be considered, therefore, as a type of "experimental neurosis." In speaking of experimental neurosis one has in mind, as a rule, the type of behavior changes studied in animals by the method of conditioned responses, introduced by Pavlov and utilized in a wide variety of experimental situations (see e.g. Masserman, 1946, pp. 122ff). In such experiments the "stress" is provided classically by presenting the animal with two conditioned stimuli between which it cannot discriminate, such as auditory tones of very small difference in frequency of vibrations, making it impossible for the animal to anticipate the correct response. According to Pavlov the consequent conflict between the excitatory and inhibitory processes results in a neurosis. The behavior manifested by the animal is similar to human behavior under conditions of severe frustration and anxiety.

Behavior disturbances may be produced also by alterations in the internal environment of the organism (McFarland *et al.*, 1941). The changes in behavior resulting from toxic factors have long been of interest to neuropsychiatrists, but rarely does one have an opportunity to study this type of stress under controlled conditions in human beings. We have reported marked effects of severe vitamin B complex restriction on personality in normal young men (Brožek, Guetzkow, and Keys, 1946); the changes were induced, as in the starvation-rehabilitation experiment, by experimental dietary restrictions and reversed by dietary treatment, and can be considered as another type of experimental neurosis.

We used the term "semi-starvation neurosis" in referring to those personality changes produced by the experimental regimen and reduced during the course of the nutritional rehabilitation which were common to all the subjects and have a parallel in natural starvation. It may be of historical interest that the term "neurosis of inanition" has been used in the past. Aronovitch in his description of the effects of the Russian famine of 1918-22, speaking specifically of children, wrote that "the neurosis of inanition manifested itself . . . in a combination of unusual vindictiveness, extraordinary irritability, and chronic crying" (quoted by Sorokin, 1942, p. 19). Yet even with this precedent we only hesitantly applied the term "neurosis" to the semi-starvation changes in the personality of the subjects. We were in a position somewhat analogous to those who have dealt with "war neuroses."

The behavioral changes, summarized by the term "semi-starvation neurosis," were universal among the subjects; hence, they may be considered as "normal" reactions under the given circumstances although they deviated markedly from the pre-starvation pattern of behavior. A similar situation is present in combat. It was recognized that under conditions of sufficient stress all men would show to some degree a failure of "normal" adaptation, the failure being evidenced by "neurotic" symptoms. "Such symptoms are . . . pathological only in a comparative sense, when contrasted with the symptoms of those still making successful adaptations" (Grinker and Spiegel, 1945, p. 7). As in starvation, "The universal stresses of combat tend to reduce all individuals to a common denominator . . . the combat personality. . . . There is a considerable repetition of symptoms, attitudes, and experiences" (*ibid.*, p. 119). This statement is not in conflict with the fact that the shared symptoms vary in their intensity from individual to individual.

In addition to the commonly shared "neurotic" symptomatology, some subjects manifested forms of behavior deviating not only from the pre-starvation condition but also from the general patterns of simple "semi-starvation neurosis." It may deserve emphasis that an attempt was made to adjust the desired weight loss in such a way that the weight decrement would take into account the "nutritional status" at the start of the semi-starvation period, and thus to equalize the physical stress inherent in the loss of weight. The subjects shared the same living quarters and were exposed to the same experimental regimen. Why did some men develop abnormal behavior patterns, going beyond the range of the common semi-starvation neurosis? Generally, one may attempt to interpret behavior differences in the light of psychophysiological constitution, past history, and situational factors. In some cases we felt able to interpret the abnormalities of behavior in these terms. In other instances the etiology and the mechanisms were not clear to us.

Psychosomatic Relationships

The behavioral, emotional, and social manifestations of starvation may be looked upon as psychosomatic phenomena, in the broad sense; that is, they are the results of a complex interaction between anatomical, physiological, individual-psychological, and social-psychological factors. In view of the psychopathological effects of vitamin deficiencies it may be noted that the diet was

relatively satisfactory as far as the vitamins, especially of the B complex, are concerned.

Behavioral changes often represented useful or necessary adjustments to physiological alterations. The decrease in body temperature was compensated for by the use of more clothing and bedcovers, by reveling in hot showers, and possibly by eating the food very hot. Because of the excessive loss of soft tissues in the buttocks, the subjects avoided sitting on hard surfaces and carried around cushions or sought out soft chairs. Physiological changes affecting the hormonal system, such as decreased activity of the sex glands (and possibly other glands), modified the intensity of the sex drive. On the other hand, the reduction of spontaneous physical activity resulted in decreasing the demands upon the energy of the organism.

In retrospect it appeared that men considered clinically as having a more stable personality showed minimal deterioration while those with latent personality weaknesses developed more severe symptoms; however, it should be acknowledged that we would have been unable to predict with any degree of certainty before the start of the stress which individuals would develop "abnormal" reactions.

The effects of dynamic psychological factors on resistance to the stress of semi-starvation deserve attention. It is evident that without a strong "superego," without firm ethical standards, none of the subjects would have been able to resist the demand of the body for food when there was plenty of it within relatively easy reach. But this general postulate throws little light on individual case histories.

We have seen the effects of attitudinal and emotional factors in an experiment (Laboratory of Physiological Hygiene, unpublished data) in which the subjects were placed on a diet practically free of thiamine after they had been maintained for some six months on low levels of thiamine intake. One subject was convinced during the first days of acute deficiency that he was receiving supplements, "felt good," and actually maintained a state of relative euphoria for nearly a week. When the depletion of thiamine stores began to reach physiologically critical values, his behavior and his mood changed in the direction of depression, as would be predicted on the basis of previous experiences (Brožek, Guetzkow, and Keys, 1946).

The temporary relief from the effects of nutritional deficiencies provided by an exciting event was observed during the siege of Breda, in Holland, in 1625 (Turnbull, 1848). In the absence of any prospect of relief from without and as a result of widespread scurvy, the besieged garrison was reduced to despair. At this time the Prince of Orange must have decided that some "psychotherapy" might be a good thing for the men in Breda. He sent letters promising speedy relief and also sent some rare medicine of supposedly great efficacy against scurvy. It was announced publicly that a few drops of the elixir were sufficient to impart a healing force to a gallon of liquor. The soldiers took the medicine with full faith in its virtue and responded excellently to the "treatment." No description of the changes in physical symptoms is given, but the emotional effects were dramatic; the feelings of despondency were replaced by a universal cheerfulness. The duration of this happy state is not known. Fortunately for the

prince's reputation, scurvy was followed shortly by a plague and the besieged garrison was obliged to surrender anyhow.

Effects of Variation in Duration and Degree of Starvation

Some of the contradictory statements in the literature on starvation may be explained by the fact that the observers referred to regimens differing in duration and degree of food reduction. The presence and intensity of hunger sensations is one of such controversial items. It appears, even though the evidence is not clear-cut, that under conditions of total starvation (no food, but water ad libitum) the subjective experience of hunger pangs disappears within a few days; Stavěl (1936) points out that even then the general emotional background, the "mood," is affected. Furthermore, under conditions of "natural" (nonexperimental) starvation the victim keeps on seeking for food, the motivation being provided not by hunger pangs but by a more fundamental drive toward preservation of life, an *élan vital*, guided in man by the knowledge that without food he will perish. In semi-starvation there is not only no reduction in the intensity of the hunger sensations but the hunger pangs tend to increase. It is only in the terminal stages that "appetite" fails.

Another, somewhat similar question is the effect of hunger and starvation on the excitability of the nervous system as judged by the response of the unconditioned reflexes. Carlson (1916) found that during hunger contractions the nervous system is in a condition of increased excitability. This fact is reflected in the increased amplitude of the patellar and other tendon reflexes. Similar observations were made by Wada (1922) in reference to muscular and mental performance. In the Minnesota Experiment, where we dealt not with hunger as a passing psychosomatic phenomenon but with semi-starvation as a physiological state, we obtained a decreased response of the tendon reflexes. This was also a common experience under the European famine conditions.

Relative Importance of the Semi-Starvation Symptoms

The question of the relative importance of the different semi-starvation symptoms may be approached by various techniques. We have used a method in which each symptom was rated by the subjects according to the degree of deterioration; the details were given in Chapter 40. In Table 384 the symptoms (complaints) are arranged in the order of severity on the basis of the average ratings of the symptoms for the whole group of 32 subjects; the rating scale for each item ranged from 0 (normal) to 5 (extreme). Increase in tiredness and appetite (desire for food) were most marked, while fainting and nausea were rated as being of negligible importance. The mental symptoms of semi-starvation deterioration were assigned a position intermediary between these two extremes.

The items included in the rating scale were selected on the basis of an analysis of the semi-starvation literature and on some a priori considerations. When the areas covered by the self-ratings were compared with the content of the interviews and the diaries, it was found that only a few points had been missed. The most important were weakness and lack of energy (which are somewhat but not adequately covered by the item "tiredness"), general slowing down, sensitivity to cold, concern with thoughts about food, and decrease in sociability. The drive

TABLE 384

AVERAGE INTENSITY OF SYMPTOMS OF DETERIORATION IN THE MINNESOTA EXPERIMENT AFTER 6 MONTHS OF SEMI-STARVATION. The items were rated on a scale extending from 0 (normal) to 5 (extreme). $N = 32$.

Symptom	Average Value	Direction of Change
Tiredness	3.5	+
Appetite	3.1	+
Muscle soreness	2.1	+
Sensitivity to noise	1.8	+
Irritability	1.8	+
Apathy	1.8	+
Hunger pain	1.8	+
Ambition	1.8	—
Concentration	1.7	—
Sex drive	1.6	—
Self-discipline	1.5	—
Mental alertness	1.5	—
Moodiness	1.5	+
Depression	1.4	+
Drive to activity	1.3	—
Muscle cramps	1.3	+
Dizziness	1.3	+
Comprehension	1.0	—
Salt craving	0.8	+
Apprehension	0.4	+
Fainting	0.3	+
Nausea	0.3	+

toward activity and the sex drive were covered by special ratings but were rated on the same scale (0 to 5) and were included in the table.

In Chapter 40 the frequencies of the different symptoms and complaints, marked by the subjects as either absent or present, were indicated. At S24, 97 per cent of the subjects tired quickly, 94 per cent felt unsteady in walking, and 81 per cent had sensations of being "weak all over." Again, the more psychological symptoms were somewhat less prominent, but they were definitely present. For instance, 66 per cent of the subjects found it hard to keep their mind on the job at hand (12 per cent at the control period), 62 per cent felt downhearted frequently (at C the percentage was 3), for 62 per cent the "guinea pig" life seemed to be a strain much of the time (at C, after 3 months on the standardization regimen, not one subject answered the question affirmatively), 62 per cent were frequently bored with people (6 per cent at C), 50 per cent preferred to be left alone (6 per cent at C), and 44 per cent became impatient when interrupted while working (6 per cent at C). On the other hand, not one subject complained of having many headaches or worried about becoming ill, only 9 per cent felt they had become more sensitive to pain, and 22 per cent thought their feelings were more easily hurt than before. It should be recalled that only 4 out of the 36 men committed serious dietary violations and in only 2 of these 4 did the reaction take a more definitely "psychotic" form.

Sorokin discussed the variability of human behavior under conditions of

TABLE 385

PROPORTIONS OF THE TOTAL POPULATION MANIFESTING VARIOUS FORMS OF DEVIATE BEHAVIOR IN STARVATION (Sorokin, 1942, p. 81).

Behavior Induced by Starvation	Percentage of the Population Succumbing to the Pressure of Starvation
Cannibalism (in non-cannibalistic societies)	Less than one third of 1 per cent
Murder of members of the family and friends	Less than 1 per cent
Murder of other members of one's group	Not more than 1 per cent
Murder of strangers who are not enemies	Not more than 2 to 5 per cent
Infliction of various bodily and other injuries on members of one's social group.	Not more than 5 to 10 per cent
Theft, larceny, robbery, forgery, and other crimes against property which have a clear- cut criminal character	Hardly more than 7 to 10 per cent
Prostitution and other highly dishonorable sex activities	Hardly more than 10 per cent
Violation of fundamental religious and moral principles	Hardly more than 10 to 20 per cent
Violation of various rules of strict honesty and fairness in pursuit of food, such as misuse of rationing cards, hoarding, and taking unfair advantage of others	From 20 to 99 per cent, depending upon the nature of the violation
Violation of less important religious, moral, juridical, conventional, and similar norms	From 50 to 99 per cent
Surrender or weakening of most of the aes- thetic activities irreconcilable with food- seeking activities	From 50 to 99 per cent
Weakening of sex activities, especially coitus	From 70 to 90 per cent during pro- longed and intense starvation

famine. Again, different effects manifest themselves in different proportions of the population, from an infinitesimal fraction on one extreme to practically all members on the other extreme. Table 385 contains the estimated percentages of the population exhibiting various forms of behavior deviating from "normal" conduct.

It is evident that even under conditions of severe famine the generally accepted codes of behavior (religious, moral, and juridical) retain to a large degree their power as codeterminants of conduct.

The Minnesota Experiment and "Natural" Starvation

In spite of the many similarities between the pictures of experimental and "natural" semi-starvation, the specific factors present in the Minnesota Experiment should not be minimized. The stress of semi-starvation was alleviated by a number of factors incidental to the controlled conditions. The subjects were provided with good physical care. They had reasonably comfortable, clean, healthful living quarters, with adequate clothing and sanitary facilities. There was plenty of soap and hot water. All the food was skillfully prepared and served. Since the subjects were not permitted to eat off diet, the danger of ingesting contaminated food or harmful substitutes was eliminated. Neither did they get the opportunity to gorge from time to time. The food was issued by the Laboratory

and there was no struggle for existence. The subjects were secure in the knowledge that their food would be served regularly. More importantly, they knew that the starvation phase would end after 6 months and that rehabilitation would follow; under conditions of natural starvation the uncertainty of the time of relief contributes greatly to the suffering of the starvation victims (for analogy among prisoners, see Farber, 1944).

The subjects in the Minnesota Experiment were under constant medical supervision and were free from political and social turmoil such as commonly accompanies famine. They could participate in the curricular and extracurricular activities at the University; they had their own educational program focused on training men for European relief; their recreational facilities were excellent. The men had strong intellectual, religious, humanitarian, and ethical values. These resources played an important role in the maintenance of morale and in keeping the men from getting into "trouble." Important sources of insecurity that are present in conditions of natural famine were absent in this experiment (e.g. bombing, threat of sudden death or injury, destruction and loss of property). Furthermore, crime, theft, depravity, exploitation, and other socially disruptive concomitants of nonexperimental semi-starvation were not present.

On the other hand, some of the restrictions imposed by the experimental regimen increased the severity of the stress as compared with that in natural starvation. It should be remembered that the subjects starved in the midst of plenty. They were unable to improve their nutritional condition by personal effort and ingenuity. The limitation of personal freedom required by the experimental program was in itself a hardship.

There was a natural, strong conflict between the desire to escape from the painful situation and the desire to continue to participate in the experiment and to gain all the satisfactions resulting from it. Although the men committed themselves at the start, there was nothing but moral pressure to keep them in the experiment. Actually the only formal penalty would have been a transfer to another Civilian Public Service Camp where they would have had food in abundance. It was a particular strain on the character of the subjects that a large measure of responsibility for conforming to the experimental regimen was placed directly on the individual himself. It may be noted that the subjects were allowed to go freely about the streets and to the homes of friends; about midway in the semi-starvation period it became necessary to establish a "buddy system" in order to lessen the strain on the individuals. But in time the buddy system became a powerful source of irritation in itself.

It is worth while to examine the hypothesis that the necessary controls and strict regimen of the experiment, aside from diet, heightened significantly the psychological stress and contributed to the behavioral changes. On this point we have only indirect though relevant evidence: 12 subjects serving on another nutritional experiment, who lived in the same quarters as the semi-starvation subjects and under similarly controlled experimental conditions, but who were on an adequate diet and were not bound by the buddy system, gave no evidence of the behavioral changes characteristic of semi-starvation. It is believed that in view of this fact the rigorous experimental regimen as such did not distort for

the group as a whole the physiologically conditioned personality changes induced by starvation.

Use of Conscientious Objectors as Experimental Subjects

Because conscientious objectors (C.O.'s) are members of a small minority group and as such are known to relatively few people except in a superficial way, some comments on the subjects as persons rather than as "guinea pigs" may be pertinent. Moreover, this may serve to forestall unwarranted criticisms of the experimental findings based on the status of the subjects as conscientious objectors.

In general, men certified as conscientious objectors by their draft boards are far from representing a homogeneous group. Some men certified as C.O.'s were actually mentally ill. There were others, such as the Jehovah's Witnesses, who were largely unwilling to accept the provisions for civilian public service and preferred imprisonment. The men selected for the Minnesota Experiment were recruited from work camps and special units administered, for the most part, by the Church of the Brethren and the American Society of Friends. The few mentally ill and the non-cooperators who found their way to these camps tended to be discharged or institutionalized. None of these was considered for inclusion in the experimental group.

Before the men were transferred to Minneapolis to serve in the experiment, they had been engaged in a variety of jobs classified as "civilian work of national importance," principally manual labor in work camps, but also in special projects where they had served as orderlies and attendants in mental hospitals, etc. More than a hundred men responded to the call for volunteers as "human guinea pigs" in the semi-starvation-rehabilitation experiment. Those candidates whose applications were approved after careful screening were physically examined and interviewed by Laboratory personnel before final selection. The men finally accepted were chosen according to these criteria: freedom from a history of disabling disease, including mental illness; absence of physical disabilities or handicaps; ability to cooperate and get along well with others; willingness to subordinate personal interests, activities, and welfare to the requirements of the experimental program; active interest in problems of nutritional relief and rehabilitation; freedom from marital or familial responsibilities.

The individuals selected were healthy, intelligent, young, white males representing a wide range of body type, physical "fitness," personality characteristics, and socioeconomic background. According to clinical evaluation as well as the results of objective measures of personality, in the standardization period only their pacifist convictions and their attitudes in some other areas of social ethics set these men apart from other men of similar age, health, intelligence, education, and background. The immature and neurotic personality trends evidenced in a few men prior to the experiment were either subclinical or mild. The men were highly regarded, both as subjects and as persons, by those with whom they worked and associated.

It seems safe to assume that any sample-bias present in the experimental findings errs on the conservative side. The psychobiological "stamina" of the

subjects was unquestionably superior to that likely to be found in any random or more generally representative sample of the population. It is unlikely that a nonselected sample of subjects could be kept to the grueling regimen for the necessary duration of such an experiment.

Social Implications of Semi-Starvation

In considering the social implications of semi-starvation it is essential to take into account the parameters characterizing restrictions of food intake. They are both qualitative (nutritional composition of food) and quantitative (rate, final degree, duration of the different phases of semi-starvation, and total duration of the food deficiency). A threat of starvation will produce different effects than the starvation itself. Also, the social effects will depend on the general economic and political organization and cultural development of the society that is threatened or affected by starvation, food being only one factor in the complicated fabric of sociological processes (see Sorokin, 1947). It is evident that generalizations arrived at by either the process of historicosociological induction or experimental analysis must be at present limited in scope.

The Minnesota Experiment was designed to explore the biology of human starvation; the social factors were held, as far as possible, "constant." Only incidentally did the experiment afford opportunities for observations of more direct relevance to the sociologist and the public administrator. The study was necessarily limited to one pattern of weight loss and, with only minor variations and adjustments, to one pattern of caloric reduction. This limits considerably the possibility of predicting behavior under conditions where the parameters of food deprivation assume other values than those present in the Minnesota Experiment. In spite of this, it seemed worth while to attempt to relate the amount of weight loss to selected, socially important effects of semi-starvation. The estimates on civil disorder and strife, capacity for physical work, and actual work performance are given in Table 386. These estimates were derived from consideration of a variety of sources, including field reports and the two experiments on semi-starvation in man (the Carnegie Nutrition Laboratory Experiment of 1918 and the Minnesota Experiment). In making these estimates it was presumed that the available food would be reasonably well distributed in the population and that the changes would take place within a period of a year. It is believed that the errors in the table will generally not be greater than ± 50 per cent of the indicated change.

The maintenance of the capacity for physical work depends upon the degree to which the work involves strength and endurance on the one hand and coordination and speed of movement on the other. The latter two aspects are strikingly more resistant to starvation deterioration than strength and, in particular, endurance. The type of work considered in Table 386 would be classified as moderately heavy. Light work, with a premium on coordination, would suffer less, and heavy physical work would be affected more pronouncedly than the table indicates. It appeared useful to differentiate between the capacity for work and the actual work performance, which may be thought of as a product of capacity and motivation.

In the Minnesota Experiment the "drive" toward activity decreased even in

TABLE 386

ESTIMATED GENERAL MAGNITUDE OF SEMI-STARVATION EFFECTS RESULTING FROM
DIFFERENT AVERAGE LOSSES OF BODY WEIGHT IN ADULT POPULATIONS
(derived from a variety of sources).

Percentage of Body Weight Lost	Magnitude of Semi-Starvation Effects		
	Civil Disorder and Strife	Capacity for Pro- longed Physical Work (%)	Actual Work Performance (%)
5.....	Slight	(?)	-10
10.....	Moderate	-10	-20
15.....	Serious	-30	-50
20.....	Very serious	-50	-80
30.....	Moderate	-80	-90
40.....	Slight	-95	-95
50.....	None	None	None

those areas in which the performance capacity remained unaltered. As we have pointed out, self-initiated mental activity decreased significantly even though the intellectual capacity remained essentially intact. The study program sponsored by the Service Committees of the Friends and the Brethren, which was designed to prepare the men for relief work abroad and which initially made participation in the experiment attractive to a considerable number of men, all but collapsed as the weight losses began to reach values of 15 to 20 per cent of the control values. The nonexperimental duties of the subjects in the Laboratory began to be carried out with manifest lack of enthusiasm and efficiency even earlier, and were still not resumed in a satisfactory way toward the end of the twelfth week of refeeding. Any effort on the part of the Staff to prop up the work program was likely to elicit a comment about "slave-driving."

Under natural conditions of inadequate food intake the decrease in the motivation to exert oneself in occupational activities may be accentuated by a number of factors. Because of a natural tendency to maintain a relatively constant body weight, a reduction in food intake (decrease in caloric input) may result in a semiautomatic reduction in work effort (caloric output); in part, the increased fatigability may serve in this case as a regulatory and protective mechanism. Furthermore, in semi-starvation and famine the consumer goods other than food are apt to be scarce also. Some authors considered this factor responsible for the low production of the miners in Great Britain in the critical years of 1946 and 1947, even in the presence of adequate diet. The stores in the mining towns were almost empty, so it did not make any real difference whether the miner worked 4 to 6 days; there was next to nothing to buy with the money anyhow.

In the rehabilitation phase of the Minnesota Experiment we were rather surprised at the slow rate of over-all recovery, including recovery in the psychological aspects of the total "fitness" of the organism. In addition to measurable (and measured) aspects of personality, such as the amount of "depression" indicated by the Minnesota Multiphasic Personality Inventory, changes were taking place in the total "social atmosphere." Management of the group was much

more difficult during rehabilitation than during either of the two other phases of the experiment (standardization and semi-starvation). Irritability seemed to increase rather than decrease. The atmosphere of aggressive hostility may be explained as resulting from frustrated expectations of a rapid restoration of normal "fitness." At times the experimenters felt as though they were watching an overheated boiler, the capacity of the safety valves remaining an unknown variable – a rather precarious situation from the point of view of "human engineering."

It may be appropriate to conclude these chapters on the psychology of starvation by citing the experience of the Allied Medical Feeding Teams engaged in relief feeding in western Holland in the spring of 1945: "The psychological condition of an underfed population constituted one of the greatest difficulties the teams encountered. The peculiar psychological state of individuals suffering from severe and prolonged calorie-shortage makes it necessary to pay the utmost attention to methods of approach, imparting of information and understanding of mental states. Apathy and irritability are the outstanding features in such a situation, which calls for special attention not only in regard to the relation between doctors and patients, but to the difficulties of dealing with civilian authorities. Good understanding will avoid delay of action and therefore be of life-saving importance. In any organization for dealing with a similar emergency in the future attention should be given from the earliest day of planning to this important aspect of undernourishment and starvation" (Burger, Drummond, and Sandstead, eds., 1948, p. 166).

Special Problems

Chapter 43. THE EDEMA PROBLEM

Chapter 44. ANOREXIA NERVOSA AND PITUITARY CACHEXIA

Chapter 45. GROWTH AND DEVELOPMENT

Chapter 46. INFECTIOUS DISEASES AND UNDERNUTRITION

Chapter 47. TUBERCULOSIS

Chapter 48. DIABETES MELLITUS AND UNDERNUTRITION

Chapter 49. CANCER AND OTHER NEOPLASMS

Chapter 50. DIETS FOR REHABILITATION, WITH SPECIAL
REFERENCE TO THE MINNESOTA EXPERIMENT

"The science in disease is medicine, and the science of the due amount in the matter of food is medicine."

ARISTOTLE, in *Nicomachean Ethics*,
Book I, Chapter 6, paragraph 4.

The Edema Problem

SINCE World War I edema has been considered an almost inevitable concomitant of severe undernutrition. It is difficult at this time to realize the extent of speculation and controversy raised in Germany in 1916 and 1917 about the "edema disease" (*Ödemkrankheit*) which appeared in the German prison camps and then spread to all parts of Central and Eastern Europe. It seems incredible that many of the German investigators did not recognize at once that simple starvation, and not some mysterious infection, was the cause. It was only after many reports from the field that the terms "hunger edema," "famine edema," and the like supplanted the first designations of "nephritis without albuminuria," "epidemic edema," "war edema," and simply "edema diseases." There were only a few vague references in the German discussions to similar conditions in previous wars, particularly the retreat of Napoleon from Moscow in 1812 (cf. Schiff, 1917b; Lewy, 1919). No one seemed to recall the experimental demonstrations that fasting organisms show water retention (e.g. Mendel and Rose, 1911).

There are many problems associated with famine edema, but the central question has to do with mechanism. What forces operate to increase the water content of the body and to maintain the edematous state? The answer can be only in part particular and limited to famine edema; at least some of the same forces must be involved in all edema, and the same general type of analysis of fluid movements and exchanges in the body should be applicable to other situations. A proper analysis of famine edema requires careful scrutiny of the factors believed to be important in edema in general.

Famine Edema before World War I

The useful information about famine or hunger edema in man prior to World War I is extraordinarily little. Potter (1904) considered the edema he saw in certain children to be the result of protein deficiency. Maver (1920) stated that the earliest account of famine edema as "essential, primary or idiopathic edema" was given by Wagner (1887). It is true that Wagner referred to "essential edema" in his title, but his paper contained no mention of famine edema or even of the edema sometimes seen in cachectic patients. Vacher (1871), under the designation of "hunger fever," reported the condition of children in the siege of Paris in 1870-71; the starvation conditions imposed by the besieging Germans resulted in progressive emaciation, edema, anemia, and diarrhea. Waddell (1826) reported a "scurvy" with edema in the starving troops in Rangoon, but the account is not clear enough to be very useful.

Some students have cited the condition of the troops in the French army that

was destroyed before Naples in 1528 as an example of famine or war edema. Hecker's (1844) account from the old chronicles, which is the only reference of value, makes it clear that the unfortunate soldiers certainly were bloated and edematous, but he makes it equally clear that serious starvation was not a major factor and that an acute contagion was involved. A better example from the same historical period is Henry the Fourth's siege of Paris in 1590; according to DeThou, the population was reduced to eating leaves and many people became hydropic (cf. Curran, 1880). Gaspard (1821) recorded the occurrence of edema in the Paris population in the famine of 1817 and also quoted a Latin chronicle of the year 586 (without stating the author), which told of the development of "bloating" in a starving population that was subsisting on grasses.

Inadequate as the old records may be, they make it clear that the misery of famine has always worn the same face. As a matter of fact, until fairly modern times famine has been of such frequent occurrence and starving persons such a common sight that it may have been considered unnecessary to describe so ordinary a subject.

General Causes and Characteristics of Famine Edema

There are a large number of immediate causes which may induce an abnormal accumulation of fluid in the body, and frequently several of these are operative at the same time. In famine, the contributing factors, besides sheer lack of calories, may be pre-existing cardiac or renal disease, dietary protein deficiency, deficiencies in one or more vitamins, protein lost in the stools from dysentery and allied conditions, anemia (especially from various parasitic infections), and excessive physical labor.

In general, the most extreme famine edema is seen where, as is often the case in India, malaria and dysentery are combined with a starvation diet and a previously bad nutritional state. With such a combination there is apt to be severe anemia and hypoproteinemia, and the edema may progress to general anasarca with ascites and hydrothorax. In less unfortunate parts of the world, such as Europe during the two world wars, famine conditions produce an edema that generally is relatively slight to moderate in clinical appearance, ascites being definitely uncommon even in the most severely starved populations. With rare exceptions the edema is of the dependent type, the affected tissues being soft, pale, and cold.

The present discussion will be confined largely to the relatively uncomplicated type of famine edema seen in Europe and in the Minnesota Experiment — that is, in the absence of infections, infestations, and a long history of vitamin deficiencies. The questions for consideration have to do with the amount of the edema, both in individuals and in populations, as well as the mechanism of its formation. As we shall see, the theories on the latter point have been rudely disturbed by recent findings. It is necessary, therefore, to examine the general concepts with some care before presenting the newer evidence.

Intracellular versus Extracellular Edema

We have discussed the morphological changes in the tissues in starvation in Chapters 9 through 14. In this chapter the question is simply as to where edema

occurs in the body, or, more particularly, whether the excessive accumulation of fluid in famine edema is confined to the extracellular space. Modern discussions of edema in general have dealt very largely with extracellular edema on the theory that edema rarely involves the cells themselves, and in any case the exchanges between cells and the interstitial fluid at present defy simple theoretical explanation. Certainly a major share of the edema fluid in famine edema, as in other common forms, is outside the cells; the ease with which the fluid mass responds to minor changes in hydrostatic pressure would seem to be proof of this. But this does not exclude the possibility that some or even a considerable amount of excess fluid may accumulate in the cells themselves.

As the cellular substance is diminished by endogenous metabolism in starvation, the cells generally diminish in size and eventually begin to degenerate. At the latter state the differentiation between intracellular and extracellular phases becomes indistinct; when the integrity of the cell wall is destroyed, even in a limited region, it must be considered that the cell has become, effectively, a part of the extracellular phase of the body. But before this happens it may be that the shrinkage in the total cell size does not correspond to the loss of protoplasm; in other words, the protoplasm may become diluted or the cell wall may enclose a watery portion as well as a more organized cytoplasmic portion. Such changes may be indicated by the appearance of vacuoles within the cell, though the full significance of such vacuoles may be argued.

Empty spaces — that is, clear, nonstaining areas with no evidence of structure — appear within voluntary muscle cells in extreme undernutrition; the appearance suggests a shrinkage of the major part of the cell away from the sarcolemma (Carville and Bochefontaine, 1875; Meyers, 1917). Cloudy swelling of cardiac muscle cells, frequently with definite vacuolization, has been reported many times (Popoff, 1885; Ochotin, 1886; Coen, 1890; Statkewitsch, 1894; Variot and Caillian, 1912; Nicolaeff, 1923). Such findings clearly suggest intracellular edema.

Similar vacuolization and cloudy swelling in gastric cells have been reported in starved salamanders (Smallwood and Rogers, 1911), in athreptic infants (Mattei, 1914), and in severely undernourished rats (Miller, 1923). The same kind of change has been observed more frequently in the livers of various starved animals and man (Statkewitsch, 1894; Stschastny, 1898; Morgulis, Howe, and Hawk, 1915; Sundwall, 1917; Asada, 1919; Krieger, 1921).

We have noted that the brain and nervous tissue in general are relatively immune to weight loss in starvation (Chapter 9). It may be significant in this connection that these tissues in starvation are particularly prone to develop vacuoles within the cells, and these have been reported by almost all investigators who have made histological examinations (e.g. Tarassewitsch, 1898; Riva, 1905, 1907; Donaggio, 1906, 1907; Agostini and Rossi, 1907; Beeli, 1908). Sundwall (1917) described swelling and vacuolization of the anterior horn cells of starved rats. It is not unreasonable to suggest that the supposed immunity of the nervous tissues to starvation loss is in part due to a replacement of cytoplasm by fluid, so that the true weight loss is masked.

Citations of histological studies like the foregoing could easily be multiplied.

The relative crudity and non-quantitative character of such observations places severe limitations on their application to any attempt at an exact analysis of the edema problem, but we are forced to the general conclusion that in starvation there is at least some intracellular edema in the sense that there are excessive accumulations of fluid within what must be considered cell walls. The question arises, then, as to whether this intracellular fluid is effectively separated from the extracellular space, the latter being defined as the space "available" to thiocyanate and other substances recently used to estimate the extracellular space. At present there is no evidence whatever on this question. If we define the total edema as the total excess concentration of water in the body as a whole, we can only say that in starvation such expedients as calculations from dilution experiments with substances like thiocyanate must result in minimal estimates.

These complications can have no place in general theories of edema at present. The probability that there is some intracellular edema in starvation must be recognized, but that is as far as it is possible to go. Subsequent discussion here, as elsewhere, must be confined to extracellular edema until better methods and information are available.

The General Theory of Edema

Since modern analyses differentiate sharply between edema and hydremia, the central problem of edema would seem to be confined to the exchanges between blood and the extravascular fluid of the body. Renal incompetence may secondarily alter these exchanges in the same way that the ingestion of water and salts can alter them, but these more remote factors must be reflected in the dynamics of the system: blood – capillary wall – interstitial fluid. Fortunately, the problem is simplified by the fact, now universally accepted, that the capillary wall is purely passive; it does not of itself push or pull fluid in either direction and does no osmotic work on the system. Moreover, the capillary wall is completely permeable to water and crystalloids; if not completely impermeable to colloids, it at least allows only minute amounts of colloids to pass through it.

Obviously, then, the major forces operating to cause fluid to move from blood to the tissue spaces will be the hydrostatic push of the intracapillary pressure and the colloid osmotic pressure pull of the interstitial fluid. The corresponding opposing forces are those of the tissue pressure, in reality the hydrostatic pressure pushing the tissue fluid against the capillary wall, and the colloid osmotic pressure of the blood plasma which tends to draw fluid to itself. Under normal circumstances, that is, in the absence of disturbances, these forces are so balanced that with the exception of the small lymph flow there is no net movement from one side to the other. A deficiency in colloid osmotic pressure in the blood plasma, however, would allow fluid to escape to the tissues, where it could only be removed by the slow flow of the lymph.

This is the theory in its elementary form as developed by Starling (1896, 1909). In more modern form the theory is conveniently expressed as an equilibrium equation:

$$(1) \quad \text{C.O.P.}_b + P_t = \text{C.O.P.}_t + P_b$$

where P_t is the hydrostatic pressure on the tissue side of the capillary, P_b is that

on the blood side (intracapillary pressure), $C.O.P._b$ is the colloid osmotic pressure of the blood plasma, and $C.O.P._t$ is that of the tissue fluid.

This equation gives quantitative description to the situation *at equilibrium*, where there is no net movement of fluid from one side to the other. Strictly, it does not consider the situation where there is a net fluid movement (represented by lymph flow) or where there is filtration in one region and reabsorption in another (pericapillary circulation).

Development of the Starling Theory of Edema

It is not surprising that these simple yet ingenious concepts were politely applauded in scientific circles and substantially ignored by clinicians for many years. In support of his theory Starling offered some crude demonstrations of the colloid osmotic pressure of blood serum but no measurements of the hydrostatic pressures. More important, perhaps, Starling provided no really critical experiments to demonstrate the quantitative interplay of these forces. Most of the facts about edema which interested clinicians were not explained, at least not directly, by the Starling theory — the mechanism of diuretic action, the important effects of salts and of acid-base balance, the role of anemia and of the lymphatics. Internists who had to deal directly with edema were more intrigued with the salt-binding theories of the French school and with the acid-swelling theory (Fischer, 1915, 1921). Leo Loeb (1923) reflected the general clinical attitude in his failure to use the Starling concept as a unifying factor in his general review of edema. Even as late as 1929 Elwyn's clinical monograph placed emphasis on a hypothetical central nervous system control of capillary permeability and disregarded more direct physical explanations of edema; Landis and Leopold (1930) sharply rejected this "vitalistic" attitude.

The critical experiment which seemed to establish the validity of the Starling theory was really supplied, or rather made effective, by World War I. While the cause of the "war edema" was being hotly debated, Denton and Kohman (1918) produced edema and polyuria in rabbits by placing them on a diet of carrots. The same effects could be reproduced by other low protein diets, and the resulting edema was more or less proportional to the lowering of the plasma protein concentration (Kohman, 1920). This emphasis on proteins coincided with the opinions of several of the writers on famine edema (Knack and Neumann, 1917; Schittenhelm and Schlecht, 1918; Park, 1918; Maver, 1920; Jansen, 1920). Confirmatory experiments on other animals were subsequently provided (Hoover, cited by Loeb, 1923; Frisch *et al.*, 1929; Shelburne and Egloff, 1931). The effectiveness of protein in the diet used to treat famine edema had been emphasized by Hoesslin (1919) among others.

But by the time the import of these points began to be clear there was no ready supply of famine edema patients to examine in Europe. In particular, there was a failure of material to study with the improved methods for the estimation of colloid osmotic pressure; these had been provided by Govaerts (1923, 1924), Schade and Claussen (1924), Verney (1926), and Krogh and Nakazawa (1927). Nephrosis patients were substituted, with results gratifying to these and other investigators (e.g. Rusznyak, 1924; Mayrs, 1926). At least some patients

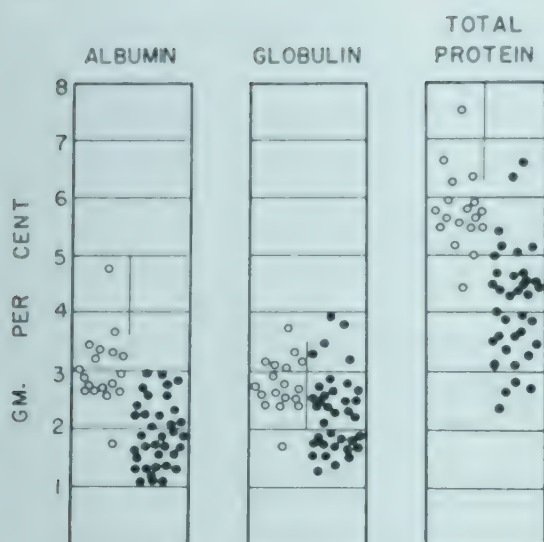


FIGURE 133. RELATIONSHIP BETWEEN PLASMA PROTEINS AND THE PRESENCE OR ABSENCE OF EDEMA; summary of 54 observations on 18 severely undernourished patients. Solid circles indicate estimations made when nutritional edema was present; open circles indicate values after the disappearance of the edema. Vertical lines indicate the "normal" range of variation as given by Moore and Van Slyke (1930). Redrawn after Weech and Ling (1931).

with nephrosis exhibited a reasonable correlation between the level of the colloid osmotic pressure of the blood plasma and the appearance or disappearance of edema.

Several clear cases of nutritional edema without renal disease were studied in China by Liu *et al.* (1931a, 1931b, 1932), who, however, measured only the total proteins. The fact that these were primarily cases of *protein* starvation may explain the success of the investigators in altering the edema at will by varying the proportion of protein in the diet. At the level of protein depletion exhibited by these patients the protein intake was obviously the limiting factor.

The studies of Weech and Ling (1931) clearly showed some relationship between plasma proteins and the presence or absence of edema in severely undernourished patients who apparently had normal hearts and kidneys. Their data from 54 observations on 18 patients are summarized in Figure 133. Obviously there was a strong tendency for edema to be present at total plasma proteins of less than 5 per cent, albumin being much more important than globulin in this respect. In their material edema did not appear if the albumin concentration was above 3 per cent but edema was usually present (in 19 out of 20 cases) when this level was under 2 per cent. At levels between 2 and 3 per cent of albumin there was little or no relationship between these variables.

In the meantime, the exposition of the Starling concept was admirably put forth by Schade and Claussen (1924) and by Krogh (1929). The differentiation in effectiveness, osmotically, between albumin and globulin explained discrepancies between total protein concentration and the colloid osmotic pressure. Bruckman, D'Esopo, and Peters (1930) and Moore and Van Slyke (1930) showed that the estimation of albumin is much more valuable than the measurement of the total protein concentration in the blood plasma.

Schade and Claussen (1924; also Schade, 1927) greatly extended the significance of the Starling theory in their picture of an outward filtration at the arterial end of the capillary, where the blood pressure is high, and a return of fluid at the venous end, where the intracapillary pressure is low. Such a pericapillary circulation undoubtedly occurs. A final major question mark about the Starling theory seemed to be answered by the direct intracapillary pressure measure-

ments of Landis (1926, 1927, 1928). These measurements indicated that the hydrostatic pressure inside the capillary was of the correct order of magnitude to balance the colloid osmotic pressure.

By the early thirties, then, the Starling theory in its more modern form was firmly established in the textbooks and in general acceptance. The edema of congestive failure was explained as the result of back pressure from an incompetent right heart, which in turn must produce increased intracapillary pressure. The edema of nephrosis was explained as due to lowered colloid osmotic pressure consequent upon albuminuria. Famine or malnutritional edema was explained in the same way as nephrosis edema except that the protein defect was on the intake side. A defect in the manufacture of plasma albumin in the liver explained hepatic edema. In general, other forms of edema were explained as the result of a loss of capillary impermeability to proteins attendant upon anoxia or local toxic actions; this would result in a decrease in the effective colloid osmotic pressure.

But few cases of simple caloric starvation had been studied and no one had attempted to apply the theory quantitatively to an unselected group of edema cases of any kind. The "proof" of adequacy of the theory rested simply on its theoretical beauty and the occasional case, usually one of nephrosis, where it seemed to correspond exactly with the facts. However, as we shall see, there remained questions of real importance and these were at least vaguely recognized by most internists (e.g. Longcope, 1934).

The Colloid Osmotic Pressure

The capillary wall readily allows the passage of crystalloids and does not act as a filter which differentiates between colloids. When any proteins "leak" through the capillary wall, the whole plasma seems to escape and the ratio between albumin and globulin in the transudate is the same as that in the blood plasma. This means that the effective colloid osmotic pressure is simply proportional to the total concentration of colloid particles, corrected for the Donnan effect. And, since the molecular weights of albumin and globulin are known — and other proteins in the blood plasma are osmotically inconsequential — this means that the colloid osmotic pressure which acts at the capillary barrier can be calculated from the concentrations of these two colloid species.

Certain deviations from the laws of ideal solutions, probably in part association phenomena, can be corrected for by empirical factors to give a nice agreement in human serums between theory and actual measurement (Keys, 1938). Various prediction equations have been proposed for estimating the colloid osmotic pressure (C.O.P.) in mm. of water, from the concentrations of albumin (A) and of globulin (G) in gm. per 100 cc. of blood serum:

- (2) C.O.P. = $75.4A + 19.5G$ (Govaerts, 1927)
- (3) C.O.P. = $68A + 21.5G$ (von Farkas, 1926)
- (4) C.O.P. = $(A + G)(21.4 + 2.9A)$ (Wells *et al.*, 1933)
- (5) C.O.P. = $f_c(45.2A + 18.8G)$ (Keys, 1938)

Equation (5), which applies to measurements near 1° C., is a least squares fit to a considerable series of normal and pathological blood serums, and it also

conforms to pure physical-chemical theory for a total protein concentration of about 3.5 gm. per 100 cc. For other concentrations ideal agreement between theory and actual measurement obtains both for natural serums and for artificial mixtures and dilutions of blood proteins when provision is made for an association factor which is dependent upon total protein concentration in the plasma. The value of f_c rises from 0.92 at a total protein concentration of 2.0 gm. per 100 cc. to 0.98 at 3.0 gm., 1.03 at 4.0, 1.09 at 5.0, 1.17 at 6.0, 1.28 at 7.0, and 1.45 at 8.0.

It is clear that the colloid osmotic pressure can be safely calculated from the concentrations of albumin and globulin in the serum as estimated by salting-out methods unless there are present considerable quantities of proteins which do not conform to the molecular sizes of these two protein species.

There have been numerous attempts to assign a definite "edema level" to the colloid osmotic pressure; below such a level edema would occur and above it there would be no edema unless an elevated venous pressure or other complications were present. Mayrs (1926) stated that in nephritis the edema disappears when the colloid osmotic pressure rises to 200–230 mm. of water. Iversen and Nakazawa (1927) concluded that edema appears when the pressure falls to 230–280 mm. Landis and Leopold (1931) reported a case of inanition edema where the edema disappeared at 235 mm. The fact is that the "edema level," even in the few cases studied, is at best a broad range. Muntwyler *et al.* (1933) observed edema in 4 nephritic patients with C.O.P. values over 200 mm. (measured at 25° C.), but the majority of their 41 patients with edema had C.O.P. values below 160 mm. Moreover, they had 8 patients with values below 200 mm. who did not have edema. In 23 cases of cirrhosis of the liver without cardiac complications, Butt, Snell, and Keys (1939) measured the C.O.P. at 1° C. They observed edema with values as high as 268 mm. of water; the lowest value found in an edema-free patient was 213 mm. Obviously, it is not possible to find a definite edema level in any one type of patient, and even the edema ranges seem to be different for nephrosis and for cirrhosis of the liver.

A corollary to the foregoing is the fact that the "normal" range of the colloid osmotic pressure is fairly wide. If all reported values for "normal" persons were considered, the range would be very wide indeed, probably from about 240 to 460 mm. of water. In many or even the majority of such reports, however, physiological standardization has been poor, so that the true range under reasonably constant conditions of rest and time after a previous meal must be narrower. In a series of 40 normal young men we have found the basal colloid osmotic pressure to range from 247 to 342 mm. with an average of 301.5. In a series of 8 similar normal young men with controlled conditions, Keys and Taylor (1935) found a range of only 283 to 321, with an average of 299 mm. of water. These values are for about 1° C. They should be increased by about 6.5 per cent for comparison with measurements made at 20° C. and by about 13 per cent for comparison with observations at 37° C.

Nishiyama (1934) made careful measurements of the colloid osmotic pressure at 20° C. of the serum in 30 normal young adults in the basal state. The range was 302 to 397 mm., the mean 330.5. Another careful study on 10 nor-

mals, with C.O.P. measured at 25° C., gave a range of 295 to 357 and a mean value of 325 mm. of water (Muntwyler *et al.*, 1933). The 5 normals studied by Fahr, Kerkhof, and Conklin (1931) showed a range of 272 to 337 mm. of water. Allowing for the differences in the temperature of measurement, all these values are fairly close to those obtained for normal persons in various studies by the present authors.

For measurements near 1° C., or for calculations using Equation (5), it is clear that the average colloid osmotic pressure of normal, moderately young adults is always close to 300 mm. of water when the subjects are not too far from the basal state. The low end of the range for such persons is around 250 mm.

The Capillary and Venous Pressures

It is not possible to measure the intracapillary pressure except by elaborate devices in a few special preparations. The direct measurement has as yet no useful application to man. Moreover, there is a sharp drop in hydrostatic pressure from the arterial to the venous end of the capillary, so that no single measurement could accurately define the total hydrodynamics in the capillary. But we can, and do, measure the limiting pressures of either side of the capillary — that is, the arterial and venous blood pressures. The arterial blood pressure seems to have little significance in edema formation, chiefly because of the large fall in the pressure which takes place in the arterioles. But the venous pressure has direct importance.

An elevated venous blood pressure means inevitably an increase in the pressure within the capillary. For convenience, we may consider only the case of the supine subject with measurement of pressure in a vein in the arm; in this case the zero point of the manometer is often taken to be 10 cm. above the table on which the subject rests. In normal persons in basal rest the pressure reads something like 10 cm. of saline solution, varying between perhaps 8 and 13 cm. When the pressure is above 20 cm. edema is almost invariably apparent, and a pressure of 17 or 18 cm. may be associated with some tendency to edema. But below 15 cm. edema is absent unless there is also marked hypoproteinemia. It is interesting that the production of edema by means of elevated venous pressure alone requires that this pressure be roughly doubled; the "margin of safety" is large.

An increase in venous pressure is the usual explanation given for the edema which forms in congestive heart failure. But Warren and Stead (1944) observed that in such patients it was generally true that the body weight and extracellular fluid volume increased while the venous pressure was normal and that the venous pressure rose *after* an increase in the plasma volume.

Until the Minnesota Experiment there had been no published measurements of the venous pressure in famine edema, although there was universal agreement that there were no clinical signs of an elevation; Knack and Neumann (1917) stated that their estimates of the venous pressure disclosed no elevation. In the Minnesota Experiment, as we have noted in Chapter 29, the venous pressure actually fell to a phenomenally low pressure. The fact that the arterial pressure was also low could only mean that the intracapillary hydrostatic pressure was considerably below the normal value. It was also observed that as recovery took

place and the edema subsided, the venous and arterial pressures returned to normal. The occurrence of edema coincided with changes in the venous pressure but in the opposite direction to that expected.

The Permeability of the Capillaries

The effective colloid osmotic pressure of the blood plasma would be reduced, of course, if some of the proteins were able to penetrate the capillary wall. The fact that protein, even though only in small concentration, always appears in edema fluid as well as in lymph has been taken by Drinker (e.g. Drinker and Yoffey, 1941) to prove that the capillaries are permeable to proteins. Further proof is claimed on the basis that when foreign proteins are injected into the blood, traces of these quickly appear in the lymph, and that the specific osmotic activity of the proteins in the lymph is much the same as that of the plasma proteins (Drinker *et al.*, 1934, 1940).

The evidence clearly proves that proteins can pass from blood to lymph, but it is important to note that there is no differential filtration in this process and that the actual amount of protein which passes across this barrier is extremely small in comparison to other exchanges. The best explanation is that there are occasional lacunae in the capillary walls which admit the passage of whole plasma. This would mean that the full osmotic effect of the proteins must be exerted everywhere at the capillary wall except where such real "breaks" occur; at these few points there could be no osmotic activity of any kind. It is entirely proper, therefore, to calculate the effective colloid osmotic pressure as exactly the same as though the capillaries were everywhere completely impermeable to the proteins. The only osmotic effect of these protein "leaks" would be exerted by the contribution to the colloid osmotic pressure of the tissue fluid of the proteins thus transferred.

The question of capillary permeability is somewhat complicated by the fact that, although there is complete permeability to crystalloids, the rate of passage of the various crystalloids and water is not uniform and unlimited. Salts, particularly bivalent ions, are able to penetrate less rapidly than water, at least under conditions of rapid transudation, and so may exert an osmotic buffering effect (Keys, 1937). While this may be important in protecting against too great and rapid departures from homeostasis in exercise and other conditions involving large pressure changes, it probably does not contribute importantly under resting conditions.

An important question is whether there is any change, particularly any increase, in the permeability of the capillary wall in starvation. The answer would seem to be in the negative in view of the composition of the edema fluid formed in this condition.

Composition of Edema Fluid

All edema fluids, and presumably all ordinary interstitial fluid, contain some protein. If there is an increased permeability of the capillaries, there should be an elevated concentration of proteins in the filtrate which passes into the pericapillary spaces and becomes edema fluid. The question is, then, What is the normal concentration of protein in such fluids (i.e., when the capillary walls are

intact and exert their full restraint against the passage of colloids)? For the establishment of this normal level one may examine situations where edema fluid may be obtained but where the integrity of the capillary wall is not seriously questioned. Cardiac failure, and venous congestion in general, would seem to represent a suitable condition.

It is widely held that an elevated intracapillary pressure corresponding to something like doubling the normal venous pressure is sufficient explanation for an accumulation of fluid in the interstitial spaces. Under such conditions excessive filtration from the capillaries would occur even in the absence of any reduction in the resistance of the capillary wall to the passage of colloids. The protein concentration in the resulting edema fluid should correspond to processes operating at a normal capillary wall. An edema fluid which does not surpass in protein concentration that formed by simple venous congestion may be considered to be formed at capillary walls which are not unduly permeable.

Analyses of edema fluid are not very numerous in the literature, and the number is much reduced when questionable methods are eliminated. At the very low protein concentrations commonly found in edema fluids, refractometric methods are open to great suspicion. The data on the composition of the subcutaneous edema fluid produced by venous congestion are reasonably consistent when only acceptable methods are considered.

In the edema fluids from 4 patients in congestive failure Falta and Quittner (1917) recorded protein concentrations of 0.13 to 0.29 per cent, and in another 4 similar patients Haas (1921) found a range of 0.03 to 0.24 per cent. Fodor and Fischer (1922) obtained corresponding values of 0.16 and 0.34 per cent, while Salvesen and Linder (1923) reported values of 0.24 to 0.35 per cent. In larger series of patients somewhat wider ranges have been reported. In 26 cardiac patients Bramkamp (1935) found protein concentrations in the subcutaneous edema fluids of 0.03 to 0.54 per cent, with an average of 0.21 per cent. In 14 such patients Stead and Warren (1944) obtained an average of 0.29 per cent (range 0.1 to 0.7).

Gilligan, Volk, and Blumgart (1934), using less acceptable methods, obtained higher values in 10 cardiac patients, 5 of whom also had diabetes; their values averaged 0.63 per cent, with a range of 0.25 to 0.91. These investigators used prolonged congestion, produced by a cuff applied for 12 hours, to obtain edema fluid from 7 normal subjects. The resulting fluid contained an average of 0.8 per cent protein, with a range of 0.7 to 1.3. Some trials with the cuff on cardiac patients also produced high concentrations of protein in the edema fluid, and it may be suspected that the cuff caused anoxic damage to the capillaries.

The foregoing evidence makes it clear that the edema fluid formed by excessive pressure on the venous side of the capillaries normally contains about 0.2 to 0.4 per cent protein, with the average approaching 0.3 per cent as a normal upper limit. At these levels and below, then, no increase in capillary permeability need be assumed. The edema fluid in undernutrition may now be considered against these reference points.

Knack and Neumann (1917) stated, without providing any data, that the edema fluid in famine edema contains less protein and nitrogen than other types of

edema fluid. Schittenhelm and Schlecht (1919) found the protein content of the fluid too low for accurate estimation with their method, but in any case it was always less than 1 per cent. Maase and Zondek (1917) analyzed edema fluid from cases of famine edema; they found the protein concentration averaged 0.116 gm. per 100 cc. (i.e., about one third as high as in parallel cases of nephrotic edema). Jansen (1920) determined the protein nitrogen in a few samples of edema fluid and found the values to be lower in famine edema than in either renal or cardiac edema. Breuer (1920) reported the value of 0.116 per cent in hunger edema. Weech, Goettsch, and Reeves (1934) produced nutritional edema in 5 dogs; the mean protein concentration in the edema fluid was 0.134 gm. per 100 cc. Youmans *et al.* (1934) reported protein concentrations of 0.37 and 0.27 gm. per 100 cc. in edema fluid from 2 patients with a mild form of nutritional edema.

For the period of World War II and after, there are surprisingly few data on the protein content of edema fluid in hunger edema. Gonnelle (1947), without providing details, stated that in his patients in Paris the values ranged from 0.15 to 0.25 per cent. In the Minnesota Experiment we found the protein concentration in casual samples of edema fluid withdrawn from the subcutaneous tissue of the ankle to contain less than 0.2 per cent protein. Govaerts (1947) studied the edema fluid in 7 semi-starved Belgians and stated that, with all allowances for the difficulties of the methods, the protein concentration is certainly something less than 0.4 per cent.

The findings of the Oxford Nutrition Survey in Holland in 1945, as reported in preliminary form (Sinclair, 1947), are at variance with all other reports. They found averages of 1.01, 0.86, 0.76, and 0.58 per cent protein with, respectively, densitometric, Kjeldahl, biuret, and gravimetric methods. Sinclair specifically stated, however, that his group would "place no reliance upon these values" and emphasized the point that in long-standing edema, with no recent accretion of edema fluid, the fluid obtained by sampling the tissues is the resultant of re-absorptive processes acting on the edema fluid originally formed. The fact is well established that edema fluid which has long been in the tissue spaces becomes very concentrated in protein (cf. Vancura, 1931; Drinker and Field, 1933).

The factors, other than capillary damage, which determine the protein concentration in edema fluid are not easy to visualize, but certain general trends are obvious. Although there is nothing like a close correlation between the concentration of proteins in the plasma and that in the edema fluid, there is some relationship; on the average the edema fluid tends to be more dilute in hypoproteinemia. There is also a tendency for the concentration to be low when the edema is in the course of rapid massive formation. When the latter situation exists on the basis of or coexistent with severe hypoproteinemia, the edema fluid may be very dilute. In a patient with ulcerative colitis studied by Landis *et al.* (1932), 6500 cc. of edema fluid was collected in 30 hours by Southey tubes. The edema fluid contained 0.09 per cent protein, and the plasma concentration was only 3.27 to 3.56 per cent, with a ratio of albumin to globulin of 0.86. The same authors reported a patient with an intra-abdominal tumor who had a venous pressure of 30 mm. Hg. The blood protein level was normal, probably owing to

repeated transfusions, and the subcutaneous edema fluid contained 0.39 per cent protein. The two patients would seem to represent, respectively, pure nutritional and pure venous congestion edemas.

Obviously there is much room for further research, but the evidence is convincing that the edema fluid formed in semi-starvation is as low in protein as the edema fluid found in any other condition. The great bulk of data indicates that it is substantially more dilute than other edema fluids. This means two things: (1) there is no evidence for any change, and certainly not an increase, in capillary permeability in semi-starvation; and (2) the colloid osmotic pressure of the tissue fluid (opposing that of the plasma) is very small. In the equation for the Starling concept the value of $C.O.P._t$ is, in hunger edema, only of the order of 10 mm. of water.

The Tissue Pressure

Of the four factors in the general equations for the Starling theory, the greatest uncertainty persists in the case of the hydrostatic pressure outside the capillary wall (i.e., the "tissue pressure" or tension). Various indirect calculations, such as those of Gildermeister and Hoffman (1922), are certainly not admissible. Smirk (1935) showed that when there is a change in the venous pressure the new equilibrium between filtering and antifiltering factors is not attained short of 5 hours, so that the calculations of Youmans *et al.* (1934) cannot be accepted. Burch and Sodeman (1937) also rejected these calculations. In this connection it may be noted that Youmans and his colleagues carried the application of the method of Landis and Gibbon (1933) farther than the originators themselves considered permissible.

The slowness with which pressure equilibration takes place in the tissues may be responsible for the great technical difficulty in obtaining satisfactory estimates of the tissue pressure by the method of direct puncture. Youmans *et al.* resorted to their indirect expedient on this account. In the Laboratory of Physiological Hygiene we have concluded that any individual measurement of the tissue pressure is exceedingly unreliable.

Subcutaneous tissue pressures obtained by the direct method have been reported for 10 normal persons by Burch and Sodeman (1937). With the heart level as the zero reference and with the point of measurement adjusted to that level, the pressures in mm. of water in different parts of the body ranged, on the average, from 17.9 (dorsum of the hand) to 37.1 (pretibial area). The general range of perhaps 10 to 40 mm. of water seems to be something like correct for normal subcutaneous tissue (cf. also Smirk, 1935). What may be the pressure in the deeper tissues, or immediately adjacent to the capillary walls, is impossible to estimate for any one moment; it may be very similar to the subcutaneous tissue pressure if sufficient time is allowed for equilibrium.

In any case, the best present evidence is that the tissue pressure is only of the order of 10 per cent as great as the colloid osmotic pressure of the plasma. The only way a change in the tissue pressure could facilitate the formation of edema would be to decrease. But the maximum decrease — a fall to zero pressure — on this reckoning could never be more than the equivalent of a decline of 10 per cent in the colloid osmotic pressure. This would appear to be trivial

and probably entirely negligible in the actual production of edema. It must be emphasized, however, that no great reliance can be placed on any current estimates of the effective tissue pressure.

The Effect of Mineral Salts and the Question of Renal Function

For many years leading French authorities – Widal, Blum, Ambard, and others – championed the theory that chloride retention is a major cause of various kinds of edema. Originally this theory was based on the proposition that the diseased kidney is unable to secrete chloride and other salts and that this would account for renal edema. In the face of evidence that this is not true (cf. Loeb *et al.*, 1932), the concept was shifted to that of protein-binding of salt in edema cases (cf. e.g. Blum and van Caulert, 1925; Ambard and Schmid, 1927). Aldrich's (1925) ideas about an increased "affinity" of the tissues for water and salt were based on his observation that subcutaneously injected saline solution disappears with unusual speed in edematous patients. But Govaerts and Bernard (1927) noted the same effect with injections of paraffin oil (see also White and Irvine-Jones, 1927).

The theories about salt and water "binding" fly in the face of analyses of the physical chemistry of the body fluids and depend, ultimately, on the simple fact that edema fluid is necessarily a salt solution which is isosmotic with a filtrate of blood. In other words, if there is much edema there must be excess salt in the body. If salt is very severely restricted, edema fluid cannot be formed and if already present will tend to be eliminated. If extra salt is given, fluid will accumulate in the starved body (cf. Jansen, 1920; Weech and Ling, 1931). These facts are not new but have recently achieved increased clinical application through the efforts of new enthusiasts such as Schemm (1942, 1944).

The argument that sodium or chloride "binding" is involved in edema has been put forth again for the case of severe undernutrition by Fiessinger and Trémolières (1943). These authors showed that in patients with severe hypoproteinemia the edema could be augmented by the ingestion of excess salt, even though there was a simultaneous increase in the protein intake and in the concentration of protein in the serum. These facts could have been readily predicted without the aid of any hypothesis about the "binding" of salt.

It is a fact that severely undernourished persons have a greatly increased hunger for salt and consume excessive quantities of water, in spite of persistent hydremia and perfectly adequate kidneys. The basic causes of these phenomena are unknown but they pose important problems. It should be noted, however, that the daily consumption of 3 or 4 liters of water and 30 to 50 gm. of NaCl does not produce edema in a normal person. Salt and water are merely the materials out of which edema fluid can be made if the mechanism for its production is operative.

In Chapters 23 and 31 we have discussed the water and salt balance and excretion in more detail. The evidence from the Minnesota Experiment, as well as from other studies (e.g. Jansen, 1920), is consistent on the following points: (1) the plasma chloride level is not particularly abnormal in any direction in cases of famine edema; (2) edema can be increased by ingesting extra salt and water and can be decreased by limiting the intake of these materials; (3) the

salt content of the edema fluid itself is not peculiar and conforms, at least roughly, to the requirements of the Donnan theory for a transudate; and (4) water and salt load tests do not disclose any abnormality of kidney response or other signs of renal insufficiency. A further indication of relatively adequate renal function is seen in the failure to find increased blood urea values.

In this connection it is essential to recognize the fact that if there were renal inadequacy this could result in hydremia, hyperchloremia, and uremia, but that the mechanism of the production of tissue edema from such alterations would still require explanation. There was actually hydremia in the Minnesota Experiment, as we have noted in Chapter 15, but the only obvious way in which this could directly cause or promote edema would be by raising the venous pressure. But the venous pressure actually fell below normal.

At the Belsen concentration camp the degree of starvation and of edema was extreme but there was no evidence for renal insufficiency. In a few cases renal function tests were made with inulin and these yielded normal values (Mollison, 1946).

World War I Theories about the "Edema Disease"

At the time of the greatest controversy about "war edema" there were no theories on edema formation, except the unnoticed one of Starling, which made any pretense of getting at fundamental mechanisms. The arguments were about the more remote causes, and these were mainly confined to two categories — infection and nutrition. In the nutrition category there were many subcategories; these were more troublesome to resolve than the question of infection which was raised at the outset but never attracted many adherents.

Rumpel (1915) first suggested that war edema might be the result of some kind of a recurrent fever, but this was emphatically denied by Hülse (1918) on the basis of a study of 145 patients. Hülse believed that war edema is secondary to nonspecific intestinal catarrh, dysentery, tuberculosis, or malaria. The frequency with which diarrhea occurred in war edema patients led to many suggestions that a dysentery was basic (cf. Kestner and Renner, 1919). The vast majority of investigators, however, quickly agreed that no infection was primarily involved. It cannot be doubted that coincidental infections frequently occur and do alter the course of the condition. Diarrhea is perhaps the most common complication, but pathogenic organisms do not seem to be involved, and the diarrhea, where it is not explained by coarse and bad food, seems to be one result of the same basic cause which produced the edema as another and rather independent result (see Chapter 26).

The nutritional causes which were proposed for war edema are still debated to some extent. They included deficiencies of calories, of proteins, of fats, and of vitamins. The suggestion that the important resemblances to wet beriberi mean a deficiency in the antiberiberi factor (thiamine) cannot be maintained in view of the small heart, the relative bradycardia in both rest and work, and the absence of changes in the nervous system. Likewise the suggestion that a kind of scurvy was involved rests on no basis whatever. Obviously, vitamin deficiencies of one kind or another frequently occur in famine, but such deficiencies are not necessary for the appearance of the "edema disease." Vitamin deficiencies were

judged definitely not to be implicated by the more critical analysts at the end of World War I (e.g. Schittenhelm and Schlecht, 1919e; Maase and Zondek, 1920).

The belief that a deficiency of fats in the diet might be causative, or at least contributory, was based entirely on the fact that the diets in World War I were, like all famine diets, low in fats (cf. Schiff, 1917b; Schittenhelm and Schlecht, 1919e). Actually, the possibility of a specific role of fat deficiency has never been fully resolved; there is no particular reason to believe that fat deficiency is important but also no real proof that it is not.

The arguments at the end of World War I that famine edema is primarily a protein deficiency disease were based simply on the small amounts of protein in the diet. Plasma or serum protein estimations in World War I were so few and so questionable that arguments based on their values were scarcely possible.

The Plasma Proteins in Famine Edema before World War II

It is easy enough to cite numerous reports of low levels of the plasma or serum proteins in cases of famine edema. Weech and Ling (1931) studied 18 cases of nutritional edema in China and never observed edema if the plasma albumin level was as high as 2.9 gm. per 100 cc., although in 2 instances the total protein concentration was above 6 gm. per 100 cc.; undoubtedly protein as well as caloric starvation was involved in their patients. Their data are given in Figure 133. It is possible to argue that the relationship between plasma proteins and edema may often be only coincidental, on the ground that in many cases there is no clear correlation between the reduction in the plasma proteins and the severity of the edema. But the most serious question is raised by the fact that numerous cases of famine edema have been reported where the plasma proteins were not particularly low.

The question should have been noted in the first sizable series of cases in which serum proteins were reported. In the 48 edematous patients studied by Schittenhelm and Schlecht (1919) the poor correlation between edema and serum proteins was obvious, but exception might be taken to the refractometric method used. Of the 40 edematous patients studied by Jansen (1920), the serum protein was below 5 gm. per 100 cc. in 4, in 16 others there were "low" values between 6.0 and 6.4 (i.e., slightly low but far from what is usually considered the edema level), and in 10 the values were fully normal—above 6.5 gm. per 100 cc.

The "epidemic dropsy" or "Bengal disease" of India does not seem to be a typical form of famine edema, though undernutrition is involved and an infectious basis has not been proved; a toxic factor derived from the consumption of oil produced from mustard seeds seems to be involved. In any case, according to Ray (1927) the total serum protein concentration is not very abnormal. In 15 cases the average was 6.49 but the albumin-globulin ratio was definitely low.

Another atypical edema in children, with no evidence of cardiac or renal abnormalities, was studied by Kostyál (1935); the results were clearly at variance with the explanation of a low colloid osmotic pressure in the plasma as the cause. Kostyál's principal findings are summarized in Table 387. It is clear that much edema was present when the plasma C.O.P. was close to or above the nor-

mal average — that is, above the so-called critical level for the production of edema on this basis alone — and that the subsidence of edema did not involve any elevation of the C.O.P. from the previous level.

TABLE 387

SERUM PROTEIN CONCENTRATION (PROT.), IN GM. PER 100 CC., AND SERUM COLLOID OSMOTIC PRESSURE (C.O.P.), IN MM. OF WATER, in 3 infants (J.N., L.N., and L.S.) with nutritional edema, in the edematous stage and when the edema had vanished or was rapidly disappearing. The C.O.P. was measured directly on the serum with the method of Kylin-Schade. (Data from Kostyál, 1935.)

Stage	J.N.		L.N.		L.S.	
	Prot.	C.O.P.	Prot.	C.O.P.	Prot.	C.O.P.
Much edema	5.47	265	7.38	380	6.53	305
Edema gone or going	6.12	250	7.85	380	5.10	290

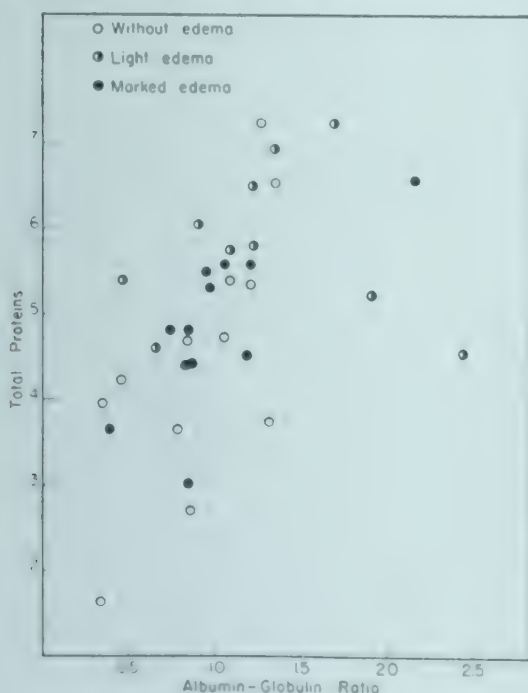
TABLE 388

BLOOD SERUM VALUES, AT THE TIME OF HOSPITAL ADMISSION, FOR 31 PATIENTS WITH ENDEMIC EDEMA. Protein concentrations in gm. per 100 cc.; C.O.P. (colloid osmotic pressure) in mm. water as calculated after Govaerts (1927) and after Keys (1938), designated as "G" and "K," respectively. (Data from Youmans *et al.*, 1933.)

Patient	Edema	Serum Protein				C.O.P.	
		Total	Alb.	Glob.	A/G	"G"	"K"
1.....	+	8.00					
2.....	+	7.50					
3.....	+	7.20					
4.....	+	7.00					
5.....	+	7.30	2.95	4.35	0.67	307	280
6.....	+	6.20	3.05	3.15	0.96	291	235
7.....	+	6.70	3.90	2.80	1.40	348	286
8.....	+	7.30	3.90	3.40	1.14	360	317
9.....	+	7.50	3.00	4.50	0.66	314	298
10.....	+	6.51	3.71	2.80	1.32	334	294
11.....	+	6.50	3.90	2.60	1.50	345	275
12.....	+	7.87	4.81	3.06	1.57	422	391
13.....	0	6.97	4.52	2.45	1.84	389	320
14.....	?	6.46	4.28	2.18	1.96	366	285
15.....	+	6.61	4.09	2.52	1.59	357	284
16.....	+	7.91	4.31	3.60	1.28	395	378
17.....	+	5.35	6.62	1.73	2.09	307	219
18.....	+	6.50	3.94	2.56	1.53	331	276
19.....	+	5.96	1.72	4.24	0.40	212	185
20.....	+	5.94	3.19	2.75	1.16	294	227
21.....	+	6.98	3.83	3.15	1.21	350	297
22.....	+	6.83	3.89	2.94	1.32	351	291
23.....	0	7.12	4.68	2.44	1.91	400	333
24.....	0	7.07	4.90	2.16	2.26	412	335
25.....	0	7.44	4.57	2.87	1.62	401	349
26.....	?	7.53	4.94	2.59	1.90	423	367
27.....	+	7.23	3.87	3.36	1.15	357	312
28.....	+	7.65	4.97	2.68	1.85	427	379
29.....	+	6.75	4.47	2.28	1.93	381	309
30.....	+	6.69	4.50	2.19	2.05	382	290
31.....	+	6.93	4.14	2.79	1.48	366	304

A more emphatic challenge to the simple hypoproteinemia theory of the origin of nutritional edema was raised by Youmans *et al.* (1932, 1933). These investigators found numerous cases of edema, with no evidence of cardiac or renal disease, in the underprivileged rural population in Tennessee. The dietary and clinical picture in these persons made it clear that general undernutrition was involved, and no other possible cause for the edematous condition could be found. Careful analyses of the blood serum, however, showed that, although both total proteins and A/G ratios were frequently somewhat low, this was not always the case, and in many instances the proteins were well within normal limits. The data on 31 of their patients are summarized in Table 388. To their values for proteins and calculations of the colloid osmotic pressure by Govaerts' (1927) method, we have added the calculation by Equation (5) above. It will be noted that the mean C.O.P. indicated by Govaerts' calculation was 344 ± 48 mm. of water, as compared with the value of 291 ± 50 from Equation (5). By comparison with normal standards for either set of calculations it is obvious that most of the values are within normal limits. With the exception of patients 6, 17, 19, and 20, all values are above the critical level at which edema may be expected on the basis of a subnormal colloid osmotic pressure. Moreover, when treatment was instituted, the behavior of the serum proteins and the edema did not correspond precisely, as they should according to the Starling theory if only the proteins were involved. Youmans and his colleagues recognized this at the time but could offer no real explanation to reconcile their findings with the hypoproteinemia theory of famine edema. On further consideration (Youmans *et al.*, 1934), they were inclined, by exclusion, to blame a lowered tissue pressure (but see above).

Opportunities to see famine edema on a large scale appeared in Spain during and following the civil war. Jiménez Diaz *et al.* (1942) saw some cases of edema with normal plasma protein concentrations and normal ratios of albumin to globulin; they also saw some with low proteins and low A/G ratios without



edema. In the most extreme edemas they usually found hypoproteinemia but concluded that certainly the edema in famine edema is not solely a result of alterations in the plasma proteins. The data from 34 of these semi-starved Spaniards are summarized in Figure 134. In this material at least, there is no evidence of any important relationship between the presence or extent of edema and the total plasma proteins, or the ratio A/G, or both.

FIGURE 134. PLASMA PROTEINS AND CLINICAL EDEMA IN FAMINE. Summary of the cases investigated by Jiménez Diaz *et al.* (1942).

Plasma Proteins and Colloid Osmotic Pressure in France, 1941-1943

In France in World War II the story was much the same as in the earlier studies. In patients with famine edema the plasma protein concentration tended to be somewhat low and the A/G ratio was often depressed, but exceptions were numerous. The conclusion of Gounelle *et al.* (1941) that hypoproteinemia was causative would be justified for some but not all of their 25 patients. A year later Gounelle and his colleagues (1942) were much less certain of the explanation in terms of hypoproteinemia and raised the question as to whether the reduction in plasma proteins, when this was found, might not be secondary to the hydre-mia.

With accumulating experience the French workers had seen that famine edema with relatively normal total serum proteins and A/G ratios is not uncommon. Relatively high values were found in the "typical cases" of Warembourg, Poiteau, and Biserte (1942) and of Vallery-Radot, Loeper, and Tabone (1943). The 6 patients of Nicaud, Rouault, and Fuchs (1942) had lost 20 to 30 per cent of their body weight but the total serum protein concentration remained above 6.0 gm. per 100 cc. in all of them. The 3 patients of Laroche, Bonpard, and Trémolières (1941) had lost 30 to 42 per cent of their body weight before they became edematous; in none of them were the plasma proteins particularly low, although the ratio A/G was depressed.

From the investigations referred to in the preceding paragraph there are adequate data for calculation of the colloid osmotic pressure by means of Equation (5), above, for 21 edematous patients at the time of hospital admission. The average calculated colloid osmotic pressure for the serum in these cases was 307 mm. of water. Only two patients ("Cr" of Vallery-Radot *et al.* and No. 2 of Laroche *et al.*) had values for the colloid osmotic pressure below 200 mm. — that is, in what might be considered the edema range.

Cachera and Barbier (1943) published valuable data on 15 patients with famine edema studied in Paris in the first years of the German occupation; in 14 the protein measurements permit calculation of the C.O.P. The data are summarized in Table 389. Many of the values for the proteins and C.O.P. are low, but for half the patients (Nos. 2, 7, 9, 11, 12, 14, and 15) the colloid osmotic pressures are above the suggested edema level.

Perhaps the most interesting feature of the French studies are the cases in which the serum proteins were studied in the same patients at various stages with and without clinical edema. The colloid osmotic pressure in the 3 patients of Laroche and his colleagues (1941) was definitely higher in the edema-free state after treatment than before. But the detailed studies of the Vallery-Radot group (1943) show some significant exceptions. Patient "Ch" exhibited 2+ edema on admission and the calculated C.O.P. was 266 mm.; several months later the edema was gone and the C.O.P. was 278 mm. (i.e., only slightly changed). Patient "Ar" had 3+ edema on admission and the C.O.P. was calculated to be 220 mm. A month later the edema was gone and the C.O.P. was 281 mm., but two weeks after this the edema reappeared and the C.O.P. was 284 mm. Patient "Ca" had 2+ edema on admission and the C.O.P. value was 234 mm., but in two weeks the C.O.P. was 257 and there was only a trace

TABLE 389

PLASMA PROTEINS AS MEASURED ON FIRST EXAMINATION IN 14 PATIENTS WITH HUNGER EDEMA. Protein in gm. per 100 cc.; C.O.P. calculated after Keys (1938). (Data from Cachera and Barbier, 1943.)

Patient	Total Proteins	Albumin	Globulin	A/G	C.O.P.
1.....	5.80	3.20	2.60	1.23	225
2.....	6.59	3.16	3.43	0.92	255
3.....	5.09	3.45	1.55	2.22	202
4.....	6.15	2.70	3.45	0.78	221
5.....	6.10	1.80	4.30	0.42	191
6.....	4.30	2.17	2.13	1.01	145
7.....	6.81	2.99	3.82	0.78	263
9.....	8.00	4.66	3.34	1.39	397
10.....	5.60	3.26	2.34	1.39	216
11.....	6.08	3.92	2.16	1.81	257
12.....	7.32	4.52	2.80	1.61	342
13.....	5.65	2.85	2.80	1.02	207
14.....	6.70	3.98	2.72	1.46	286
15.....	7.65	4.29	3.36	1.27	352

of edema; this questionable edema state continued for several more weeks while the C.O.P. rose to 291 mm. of water. But several months later the edema reappeared in patient "Ca" although the C.O.P. was then 382 mm. Similar failures of correspondence between C.O.P. and the course of the edema appear in the data of Cachera and Barbier (1943).

Other investigators in France questioned the explanation of famine edema as caused by hypoproteinemia and lowered colloid osmotic pressure (cf. Loeper, Varay, and Mende, 1942). Bachet (1945) observed that some rough correspondence between the presence of edema and the level of the plasma proteins may be seen in mixed groups of undernourished persons, but when serial studies are made on individuals the picture is especially informative. In the men carefully studied in a mental hospital it was found that the edema often appeared first, and only after it was established was there a fall in the plasma proteins. Certainly any review of the French work would seem to dispose of the C.O.P. theory as the sole or even the major factor involved.

Famine Edema in Belgium and Holland – World War II

Famine edema was not very common in Belgium in World War II, but at the St. Pierre Hospital in Brussels 159 cases of edema were seen in 1941 and 188 in 1942. A few cases were seen at Liège by Brull and his group, who carried on studies on nutrition all through the period of German occupation (Brull, ed., 1945). Brull and his colleagues agreed that hypoproteinemia and a low colloid osmotic pressure are factors in famine edema but that "Renal insufficiency and consequent water retention with a normal or overnormal water intake is primarily responsible for the appearance of hunger edema" (p. 121). Their belief in the role of renal insufficiency seems to have been based entirely on the results of some perfusion experiments with dogs which at best have only a remote bearing on the problem.

When Belgium was liberated, the entering medical officers conferred with the leading Belgian authorities to get their theories about the causation of famine edema. Professor Govaerts ascribed it simply to hypoproteinemia. Professor Brull blamed the edema on renal insufficiency. And Dr. Simonart considered it to be a result of thiamine deficiency. Govaerts' data are discussed in a separate section below.

Famine edema was very common in the disastrous winter and spring of 1945 in the Netherlands. A variety of data were collected on the incidence of edema and the level of the plasma and serum proteins. In general, individual cases were not studied in detail but some of the data are suggestive. In Amsterdam and Utrecht in May 1945 it was found that random samples of the plasma proteins in the street population gave values never less than 5.8 gm. of total protein per 100 cc. of serum and that averages for both men and women and for various economic classes were always above 7.0 gm. per 100 cc. But 4.2 per cent of this street population showed famine edema at the time of the examination. In Amsterdam and Leyden 230 serum analyses were made without finding a single abnormally low value.

Professor Jansen and Dr. Spaander studied a few patients in Amsterdam in May and June of 1945. Some of these persons were free from edema although the serum proteins were very low. For example, their patients numbered 18 and 19 had, respectively, total protein concentrations of 3.8 and 3.7 gm. per 100 cc. and A/G ratios of 0.9 and 1.05, but clinical edema was not present. These values correspond to colloid osmotic pressures of 118 and 119 mm. of water, or 8.7 and 8.8 mm. of mercury. On the other hand, of the 4 patients who had clinical edema, one (No. 29), whose edema was graded 2+, had 6.1 gm. of total proteins, an A/G ratio of 1.35, and a C.O.P. of 265 mm. of water. After ten days this patient was free of edema but the total serum protein concentration was then 6.0 gm. per 100 cc., the A/G ratio 1.22, and the C.O.P. 254 mm.

Govaerts' Data from Belgium, 1941

Govaerts (1947) has reported a series of observations, including estimates of the colloid osmotic pressure of the blood plasma, which merit detailed examination. The observations concern edematous patients drawn from the underprivileged population of Brussels. Most of these patients were studied in 1941 but a few apparently were seen in 1942. This was a period of food shortage, but certainly nothing approaching famine. Govaerts stated that his patients had been eating not more than 1500 Cal. daily with an average of 25 gm. of protein, practically none of which was animal protein. In the absence of any details on body weight it is impossible to estimate their actual nutritional status, but there is no reason to doubt that they were, in general, at least moderately undernourished with regard to calories and severely undernourished with regard to proteins.

The data of moment for the present discussion are summarized in Table 390. Several features should be noted at the outset. They were, with few exceptions, relatively old people, averaging 54.8 years of age. In general, the basal metabolic rates and pulse rates were at the levels associated with moderate to severe caloric undernutrition, but the blood pressures were far from characteristic. Only 6 of the group had blood pressures even approaching the famine level, not one

TABLE 390

DATA FROM CASES OF EDEMA IN BELGIUM IN THE YEARS 1941-42. C.O.P. = colloid osmotic pressure in mm. of water. Protein = total serum protein concentration in gm. per 100 cc. Blood pressures are expressed in mm. of mercury. Circ. Time = arm-to-tongue circulation time in seconds. B.M.R. = metabolic rate as percentage deviation from normal (standard not given). (Govaerts, 1947.)

Patient	Sex	Age	Edema	C.O.P.	Protein	Blood Pressures		Circ. Time	B.M.R.	Pulse Rate
						Arterial	Venous			
2.....	M	64	+++	130	3.68	105/70		16		64
3.....	F	50	+++	135	4.18	160/90	12	24	-36	70
4.....	M	53	+++	140	4.18	150/70	9	18	-60	64
5.....	M	55	+++	150	4.40	160/100	5	18	-32	48
6.....	M	70	++	160	4.60	150/90	20	20	-38	50
8.....	M	42	++	165	4.39	100/70	12	24	-24	52
9.....	M	77	++	170	4.16	100/50	13	20	-43	80
12.....	M	41	+	185	4.73	140/90	10	16	+10	54
13.....	M	55	+	185	5.24	130/100	12	24	+8	60
14.....	M	50	+++	185	5.68	120/90	15	20	0	88
15.....	M	59	+++	195	5.18	140/70	12	27	-25	50
17.....	M	81	+	205	5.65	130/70	11	22	-28	52
18.....	M	63	+	215	5.43	150/90	9	22		55
19.....	M	57	+	220	5.90	150/70	10	22		48
20.....	M	68	++	220	6.27	170/80	5	27	-20	38
21.....	M	59	+++	223	5.52	150/80	6	18	-42	60
22.....	M	43	+	225	6.01	130/70	7	18	-22	55
23.....	F	32	++	225	5.99	110/70	8	20	-24	54
25.....	M	64	+	235	6.55	130/70	5	22	-42	74
26.....	M	73	+	240	5.79	150/80	10	20		45
27.....	M	52	++	245	6.38	130/70	10	22	-18	58
28.....	M	68	++	257	5.84	140/70	9	20	-20	57
29.....	M	34	+	275	6.77	125/60	12	19	-22	44
30.....	F	43	+	275	6.93	135/75	10	18		60
31.....	M	49	++	290	7.35	130/70	6	20	-23	54
32.....	M	51	++	290	6.10	170/90	11	22		48
33.....	F	52	+	290	5.86	130/75	13	23		52
34.....	M	54	++	290	6.53	120/80	14	22	-54	40
35.....	M	32	+	295	6.77	105/70	7	19	-28	56
36.....	M	35	++	325	7.42	120/80	7	19	-14	44
38.....	M	72	+	350	5.47	110/70	11	20	-46	58
M.....		54.8		225		134/75				

person had hypotension, and 13 of them would qualify as at least moderate hypertensives according to our standards for well-fed persons. At least 10 were markedly hypoproteinemic. The venous blood pressure in 21 out of 32 was above the starvation maximum recorded in the men of the Minnesota Experiment (see Chapter 28). Compared with the Minnesota subjects, Govaerts' patients were much older, had more severe edema, had higher pulse rates and metabolic rates, were more hypoproteinemic, and had far higher arterial blood pressures.

The greatest interest in Govaerts' series attaches to the colloid osmotic pressures. These are undoubtedly low in most of the cases, and Govaerts laid stress on the fact that, according to his calculations, the C.O.P. was usually less than would correspond with the estimated concentrations of albumin and globulin in the serum. Unfortunately, the data are not given to check this calculation, but

it is stated that it was made with the equation previously developed by Govaerts (1924, 1927): $C.O.P. = 75.4A + 19.5G$, where C.O.P. is in mm. of water and A and G are the concentrations of albumin and globulin, respectively, in gm. per 100 cc. of serum. This equation is erroneous in at least two respects (cf. Wells *et al.*, 1933; Keys, 1938). In the first place, it does not allow for the well-established fact that the specific colloid osmotic pressure of the plasma proteins rises with increasing concentration, so that, for example, the C.O.P. per gm. is 25 per cent greater at a concentration of 7.0 gm. per 100 cc. than at 4.0 gm. per 100 cc. (Adair, 1925; Meyer, 1932; Wells *et al.*, 1933; Keys, 1938). Secondly, Govaerts' equation indicates that the mean molecular weight of the plasma globulins is almost 4 times that of the albumin instead of the established fact that the ratio is 2.4 (Keys, 1938). No particular weight, therefore, can be placed on the alleged discrepancy between these measurements of C.O.P. and the calculations from "theory."

Govaerts (1941) indicated that the average normal C.O.P. by his method is about 405 mm. of water, and he found that all his measured values in these edematous patients were considerably below this level. Actually, his mean "normal" value of 405 mm. is significantly higher than accepted elsewhere; a better figure would be about 310 for the mean, with a lower normal extreme at about 250 mm. (cf. Meyer, 1932; Butt and Keys, 1939). It will be noted that 10 of the 31 C.O.P. measurements in Table 390 are, in fact, within the range of values actually found in normal persons in other investigations.

For 5 patients Govaerts (1947) reported values with edema and after its

TABLE 391

DATA ON 5 PATIENTS DURING FAMINE EDEMA AND AFTER DISAPPEARANCE OF THE EDEMA. C.O.P. = colloid osmotic pressure in mm. of water. Protein = plasma protein in gm. per 100 cc. Circ. Time = arm-to-tongue circulation time in seconds. Blood pressures are expressed in mm. of mercury. B.M.R. = metabolic rate as percentage deviation from normal (standard not given). (Govaerts, 1947.)

Patient	C.O.P.	Protein	Circ. Time	Blood Pressures		B.M.R.	Pulse Rate
				Arterial	Venous		
De (Male, 54 yrs.)							
Edema	290	6.53	22	120/80	14	—54	40
Edema-free	240	6.94	18	130/80	6	—41	48
Tu (Female, 32 yrs.)							
Edema	225	5.99	20	110/70	8	—24	54
Edema-free	325	8.21	16	120/80	8	—15	76
Cl (Male, 49 yrs.)							
Edema	290	7.35	18	130/70	6	—23	54
Edema-free	280	7.42	15	140/70	8	—13	69
Ri (Male, 68 yrs.)							
Edema	220	6.27	27	170/80	5	—29	38
Edema-free	260	7.35	20	170/90	9	—18	50
Le (Male, 68 yrs.)							
Edema	257		20	140/70	9	—20	68
Edema-free	293		14		7	—14	75
M							
With edema	256.4	6.55	21.4	133/75	8.4	—32.2	50.8
Without edema	279.6	7.48	16.6	143/80	7.6	—20.2	63.6

disappearance. These are summarized in Table 391. In 2 out of 5 patients the C.O.P. was *lower* in the edema-free state. Neither during nor after edema were the plasma proteins low in any of these 5 patients.

Govaerts gave the major role in the production of edema in his patients to a lowered C.O.P. and ascribed part of this lowering to some unknown change in the blood proteins. But he also recognized the complexity of the situation and mentioned muscle tonus, transmitted pulsatile pressure, and turgor as influential factors. His own data show that C.O.P. is by no means the only determinant and that in a number of his patients edema was present in the face of relatively normal C.O.P. and venous pressures.

Oxford Nutrition Survey—World War II

Sinclair (1947) provided an outline of the observations made by the Oxford Nutrition Survey and various British and Dutch associates in Holland and Germany at the end of World War II and shortly thereafter. Between September 1945 and July 1946 a total of 62,254 German subjects were examined, of whom 4240 (6.8 per cent) had famine edema, 4860 (7.8 per cent) had edema judged to be probably not nutritional in origin, and the remainder (53,154) showed no clinical edema. From the dietary histories it was concluded that famine edema may arise in German adults with intakes of 600 to 1800 Cal. per day.

Table 392 summarizes Sinclair's data on concentrations of total proteins and of albumin in the blood serum. All the protein values are high and, as averages, exceed most acceptable averages for normal men and women determined elsewhere. Since bloods were apparently drawn under non-basal, non-resting conditions, and the methods were those adaptable to field use, the results are not fully comparable with normal standards, but it is clear that on the average there was no hypoproteinemia or hypoalbuminemia in these groups. On even firmer

TABLE 392

MEAN VALUES FOR TOTAL PROTEIN AND FOR ALBUMIN IN THE BLOOD SERUM OF GERMAN ADULTS WITH FAMINE EDEMA, WITH EDEMA OF NONNUTRITIONAL ORIGIN, AND WITHOUT EDEMA. X Prot. = densitometric estimation; Y Prot. = biuret method; Alb. = albumin estimated by the biuret procedure. All values are in gm. per 100 cc. of serum. (Sinclair, 1947.)

Condition	Males					
	X Prot.		Y Prot.		Alb.	
	N	M	N	M	N	M
Famine edema	76	7.6	26	7.5	25	5.0
Other edema	61	7.6	37	7.8	41	5.1
No edema	1584	7.8	653	7.7	694	5.0

Condition	Females					
	X Prot.		Y Prot.		Alb.	
	N	M	N	M	N	M
Famine edema	67	7.8	29	7.9	31	5.1
Other edema	201	7.8	139	7.9	140	5.1
No edema	1385	7.8	781	7.8	780	5.1

ground is the comparison between the persons who had edema and those who were edema-free. The presence or absence of edema was independent of the protein and albumin levels in the serum.

In connection with the Oxford Survey, measurements of the colloid osmotic pressure were made on the blood serum from 13 men with famine edema. The data are summarized in Table 393. None of the C.O.P. values was within the usual range associated with edema from lowered C.O.P., and most of the values were well within the usual range for normal persons although they were below the usual *averages* for groups of normal men. Sinclair stated (p. 85) that the C.O.P. was low but that the albumin and globulin values were normal. It may be true that the C.O.P. was lower than would be estimated from the protein analyses, but the requisite details are not available to check the calculation; we have noted already that Govaerts' equation is inapplicable, and this may have been used by the Oxford Survey. But in any case the fact remains that the C.O.P. values themselves are not low enough to explain the development of edema.

TABLE 393

COLLOID OSMOTIC PRESSURE, IN MM. OF WATER, AND TOTAL PROTEIN CONCENTRATION, IN GM. PER 100 CC., IN THE BLOOD SERUM OF 13 MEN WITH NUTRITIONAL EDEMA in western Germany after World War II. Mean protein = 7.21; mean C.O.P. = 290.8. (Sinclair, 1947.)

	Case Number												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Protein ...	6.3	6.8	7.0	7.0	7.0	7.2	7.2	7.2	7.2	7.3	7.3	7.4	8.9
C.O.P.	263	290	280	292	300	248	270	290	298	290	300	305	355

Sinclair expressed the belief of the Oxford Survey that "the lowered colloid osmotic pressure plays only a small part in the cause of the edema" (*ibid.*, p. 91) and pointed out that, accordingly, "other factors must be sought." Sinclair did not examine the basic theories of edema but did conclude that in Holland and Germany the famine edema of World War II could not be ascribed to cardiac failure or to thiamine deficiency. His final point, that "the role of the kidney needs further investigation," is justified, but he expressed doubt that the kidney is the most important factor.

The Warsaw Ghetto—World War II

From the end of 1939 until final destruction in the early summer of 1942, the inhabitants of the Warsaw Ghetto underwent the utmost food deprivation. As elsewhere under such conditions, edema became commonplace; eventually everyone became classifiable as having either famine edema or dry cachexia (see Apfelbaum, ed., 1946). For the entire period the autopsy records showed that 32.5 per cent of the victims were edematous, but this may not correspond accurately with the actual percentage of edema at death. Although a total of 3658 autopsies were done, the post-mortem examinations could by no means keep up with the death rate; in the hospital the daily mortality rate reached 20 to 30 per cent

(Stein and Fenigstein, 1946). Of the edematous bodies the pathologists recorded that about half of them were hydropic (anasarca) and 13.8 per cent of them exhibited a brownish pigmentation of the skin.

Among the children edema was most marked between the ages of 2 and 5 years, and when it developed at this age it was considered a grave prognostic sign (Braude-Heller *et al.*, 1946). With advancing age, edema was considered to be less serious in portent, and in adults the majority of deaths occurred in the cases of "dry" cachexia (Stein and Fenigstein, 1946). The edema was generally of the dependent, symmetrical type. Unsymmetrical edema was associated with thrombosis of the femoral or iliac vein, and this complication was not infrequent in Warsaw (Braude-Heller *et al.*, 1946).

The Warsaw investigators were impressed with theories of "hydrophylia" of the tissues and attempted to estimate this with the McClure-Aldrich test, in which saline is injected subcutaneously and the time is measured for the wheal to disappear. Whereas the disappearance time was taken to be about 60 minutes in normal persons, the time was greatly shortened in the Warsaw famine victims. In 40 adults and 20 children the disappearance time was usually 20 to 30 minutes and in some cases was as low as 15 to 20 minutes.

Various other special studies were carried out at Warsaw in an effort to explain and characterize the famine edema. Attempts to study water balance only showed that famine victims have a high rate of water turnover associated with thirst and an elevated fluid intake; the extrarenal water output was normal except in cases of diarrhea (Fliederbaum *et al.*, 1946). A few measurements were made on the venous pressure in cachectic and in well-nourished young adults at rest and after work; there was no essential difference in the average results from the 2 groups, but among 7 cachectic persons 3 had slightly subnormal venous pressures and 2 had moderately elevated values (Apfelbaum, ed., 1946).

Edema Observed at Mainau, 1945

Many starving men released from German concentration camps were observed at Mainau by Lamy, Lamotte, and Lamotte-Barrillon (1946, 1948), who selected for special study a group of patients in whom the complications of other disease were not immediately apparent. The general applicability of the findings is, however, rendered questionable by several peculiarities of this group as contrasted with the majority of other starving persons who have been examined. At Mainau tachycardia was the rule instead of the usual starvation bradycardia; the resting pulse was generally between 95 and 120. The venous pressure was in the upper range of normal and both venous pressure and pulse rate rose rapidly with slight effort. Of 28 patients, 17 were febrile and only one man showed the usual hypothermia. Lamy and his colleagues (1948) also reported evidence for renal damage.

It would appear, then, that the patients at Mainau were on the verge of cardiac and renal failure, as well as, in most cases, probably suffering from febrile infections; the problem of edema in that group seems to be rather different than in most other famine experience. The evidence on the kidney is discussed in Chapter 31.

The greatest interest attaches to the studies by Lamy, Lamotte, and Lamotte-

Barrillon (1946, 1948) on the colloid osmotic pressure of the serum proteins in 10 undernourished men, 5 of whom had marked edema. The first point that emerges from this is that the colloid osmotic pressure was definitely low in the serums drawn from the more edematous men. Comparison of the osmotic pressures with the estimated protein concentrations in the serum led Lamy and his collaborators to conclude that the osmotic pressure per unit of protein was also subnormal. Precise evaluation, however, is difficult if not impossible in view of the very high levels of protein concentration reported. In the 10 patients the reported total protein concentrations in the serum ranged from 5.90 to 10.50, with a mean of 7.73 gm. per 100 cc.

Such extraordinarily high protein concentrations presumably mean that either the patients or the methods, or both, were peculiar. Obviously, the Mainau data cannot be compared directly with the findings elsewhere and therefore do not in themselves afford a basis for any general conclusion. Lamy, Lamotte, and Lamotte-Barrillon (1948) also made ultracentrifugal studies on 5 serum samples from 2 edematous patients, which they suggested could explain the discrepancy between their osmometric values and the protein determinations. They obtained evidence for the presence in the blood serum of a heavy particular, of the order of 1,000,000 in molecular weight, which constituted less than 10 per cent of the total serum protein mass. This is extremely interesting but does not support the contention that the total osmotic activity of the serum proteins is markedly aberrant. By simple calculation it would appear that the presence of 10 per cent or less of the total protein in the form of a molecule of weight 1,000,000, in serum where the ratio of albumin to globulin is of the order of 1.5 (as in the Mainau men), would result in a reduction of only 5 per cent in the colloid osmotic pressure as compared with the expectation for serum not containing such an abnormal fraction.

From a detailed examination of the literature and their own data, Lamy and his colleagues (1948) concluded that a number of factors combine to explain the development and maintenance of famine edema. Besides a dominant tendency to low colloid osmotic pressure of the serum, related to low protein concentration and a low osmotic activity per unit of protein, they recognized circulatory deficiencies, hydremia, and an augmented blood volume. They considered that factors other than colloid osmotic pressure are relatively more important during rehabilitation and in cases of relatively moderate undernutrition.

The complexities of the problem of famine edema are not lessened by the evidence from Mainau; new questions are posed for future studies. In any case, the Mainau data do not directly pertain, without much more evidence, to the ordinary starvation situation in which there is bradycardia, low venous pressure, and no albuminuria. It is clearly necessary, however, to explore further the osmotic activity and the physical chemistry of the plasma proteins in patients with famine edema.

Lamy, Lamotte, and Lamotte-Barrillon (1948) properly point out that the evidence of dehydration in undernutrition deserves far more attention than has as yet been accorded it. Not only are there apt to be more cases of dry cachexia than of famine edema, but even the edematous cases show signs of dehydration in some respects. Excessive thirst, desiccated skin, and extremely dry mucous

membranes have been noted by many investigators besides the Mainau group. It should be noted that excessive thirst appears in other forms of edema besides that resulting from semi-starvation (Schemm, 1944). Lamy and his group suggest that the cells are dehydrated by losing water to the extracellular spaces and that this accounts for the elevated temperature they saw in many starving persons. We have already noted, however, that the presence of hyperthermia in the Mainau patients is one of the surest indications that these were not cases of uncomplicated starvation.

Simonart's Theory of Thiamine Deficiency

Simonart's book (1948) on wartime undernutrition is largely devoted to expounding his theory that famine edema, at least as seen in a Belgian civil prison in World War II, is simply a form of wet beriberi. The men studied by Simonart were certainly undernourished, some of them had very marked edema, and they exhibited many of the classical signs of simple semi-starvation. However, there are some indications that these men were not typical of simple undernutrition, particularly in the excessive edema for the degree of emaciation. Simonart's arguments for a thiamine deficiency would be tedious to recapitulate here; some of them, such as the heart rate, actually argue against his theory, and others, such as the data on pyruvic acid and muscle fatigability, are clearly based on faulty technical methods. The relative absence of real signs of polyneuritis speaks strongly against a prominent degree of thiamine deficiency in his subjects. It is significant that Simonart made no thiamine analyses on either the food or the excreta of his patients. The only evidence of consequence is the reported beneficial results of thiamine administration.

Simonart gave intravenous injections of thiamine to 6 patients and reported that, without any other treatment, the serum protein level rose in 5 of these men and that there was an increase in the ratio of albumin to globulin. Moreover, Simonart suggested, without evidence, that thiamine has some direct influence on the proteins so as to increase their osmotic activity. In some cases a dramatic diuresis was reported to have developed following thiamine treatment, but it is not clear whether or not there were other factors involved, such as bed rest and extra food. It was stated that in one or two patients enlarged hearts diminished in size after thiamine.

It is impossible to decide whether or not there was any real thiamine deficiency in Simonart's patients. If there was, this seems to be almost unique for the semi-starvation seen in Europe in World War II. Elsewhere in Belgium, studies on thiamine metabolism in undernourished persons gave no indications of deficiency (Lambrecht *et al.*, in Brull *et al.*, 1945). In France, in the Netherlands, in western Germany, in other groups in Belgium, and in the concentration camp victims seen at Mainau and in Switzerland, the findings, including vitamin analyses, did not indicate any significant thiamine deficiency (Hoogland, 1947; Steijling, 1947; Dumont, 1945; Sinclair, 1947; Gsell, 1948; Lamy *et al.*, 1948). It does not appear that the consumption of a calorically deficient diet composed of dark bread, potatoes, and the common vegetables of Northern Europe is productive of signs attributable to a lack of thiamine—and indeed, such a diet, contrary to Simonart's claim, is not low in this vitamin.

The reports from a very considerable number of trials with thiamine and polyvitamin treatment in European famine edema fail to support Simonart (Gounelle *et al.*, 1942, 1943; Sinclair, 1947; Dumont, 1947; Lamy *et al.*, 1948). Similar edema in the Far East likewise failed entirely to respond to thiamine (Kurnick, 1948). Bachet (1943) used large doses of thiamine for many days in treating edematous patients in a mental hospital and found no effect on clinical edema, body weight, or plasma proteins. Thiamine alone did not affect the edema of the undernourished miners on the Island of Elba (di Granati *et al.*, 1947).

The Studies of Beattie, Herbert, and Bell, 1945-1946

The studies of Beattie, Herbert, and Bell (1948) on edema in severely undernourished Dutch (1945) and German (1946) adult males are of particular value because of their maintenance of controlled conditions, the elimination of secondary complications, the use of precise methods, and, above all, the clear appreciation of the problems involved. Such matters as the collection of blood without stasis and under basal conditions, the specification of all methods, and the use of modern statistical methods are almost unique in the study of famine victims.

Table 394 shows the data on the degree of clinical edema, the plasma proteins, and the calculated colloid osmotic pressure for the 20 patients on whom the requisite data were obtained. It is important to note that these data refer to the men before any appreciable treatment was instituted. It is obvious that in these Dutch victims of the famine of 1945 there was no close relationship between the level of the proteins in the plasma and the degree of edema. The two patients, O. and F., who had the severest edema also presented low plasma proteins and subnormal values for the calculated colloid osmotic pressure, and in these two individuals it is reasonable to suggest a causal relationship. But in the other 18 persons there is substantially no apparent relationship.

The data for the Germans studied by Beattie and his colleagues are insufficient for the calculation of the colloid osmotic pressure, but it is of interest to note the total protein concentrations in the plasmas of the 11 men studied before and after limited refeeding. Eight of these 11 men were edematous before refeeding; none was edematous afterward. The before and after mean total plasma protein concentrations were 6.66 (range 6.02 to 7.21) and 6.89 (range 6.36 to 7.22) gm. per cc., respectively.

Beattie, Herbert, and Bell estimated the plasma and extracellular fluid volumes in the Germans by the dye (T1824) and thiocyanate methods; in the Dutch patients only the plasma volume was estimated. The data on plasma volume are summarized in Table 395. The mean absolute plasma volume was apparently unchanged by starvation in both groups, when the pre-starvation value is calculated as 45.3 cc. per kg. of "normal" weight as found for normal young men by Henschel *et al.* (1947). The relative plasma volume per unit of body mass was, therefore, expanded in proportion to the degree of weight lost. Considering only the observations made before refeeding began, it appears that the Dutch patients had an average plasma volume of 70.6 cc. per kg. of their body weight at that time; compared with the normal average of 45.3 cc. per kg.,

TABLE 394

PLASMA (OR SERUM) PROTEINS IN SEMI-STARVATION, recorded in gm. per 100 cc., by Beattie, Herbert, and Bell (1948). The colloid osmotic pressure (C.O.P.), in mm. of water, is calculated after Keys (1938). Grading of the edema: 0, no edema; 1, no visible edema but pitting edema limited to the foot and ankle region; 2, visible edema of ankle and foot; 3, edema extending to the knee; 4, edema to the inguinal ligament; M, massive edema of all four limbs and trunk.

Subject	Sex	Age	Weight Loss (%)	Edema	Total Proteins	Albu- min	A : G	C.O.P.
D.*	F	53	49	0	6.25	3.18	1.04	240
V.A.*	M	57	40	0	6.54	3.66	1.28	267
T.Sr.	M	52		0	6.75	3.46	1.05	275
d.G.*	M	58	29	0	6.73	4.64	2.22	314
V.*	F	66	38	0	6.88	4.46	1.84	314
T.Jr.*	M	21		1	7.55	4.61	1.57	356
Bg.*	M	74		1	6.62	4.56	2.21	301
Z.*	F	62	36	1	6.52	4.05	1.64	280
E.*	M	36	27	1	7.23	4.46	1.61	332
Sp.	F	54		2	6.90	3.90	1.30	296
Vr.	F	62	47	2	6.27	3.90	1.65	263
Fr.*	F	44	41	2	5.91	3.32	1.28	237
T.*	F	55	37	2	5.25	3.16	1.51	202
B.	M	45		2	7.10	3.27	0.85	271
J.*	M	59	29	2	6.50	3.35	1.06	257
Zn.	M	48		3	7.78	4.54	1.41	373
V.V.	F	52	33	3	6.28	3.56	1.31	252
W.*	F	62		3	6.70	4.34	1.84	301
O.*	F	53		4	5.44	3.80	2.32	221
F.*	M	77		M	4.60	2.09	0.83	150
M								
Edema, 0 to 1..					6.78	4.12	1.61	297
Edema, 2 to 3..					6.25	3.57	1.40	257

* These analyses were made on serum.

this indicates an average excess of 25.4 cc. per kg. for these persons, who then averaged only 62 per cent of the "normal" weights for their heights and ages. In the Germans, who averaged 25 per cent below normal average weight, the mean plasma volume amounted to 62.4 cc. per kg., or an average excess of 17.1 cc. per kg.

The findings for the thiocyanate space on the Germans are summarized in Table 396. Before refeeding, the thiocyanate space averaged 34.2 (*SD* = 1.43) per cent of the actual body weight at the time. This may be compared with the mean value of 23.5 per cent obtained by Henschel *et al.* (1947) for normal men. The absolute thiocyanate space, however, was only slightly expanded from the pre-starvation level if this latter is estimated at the normal level for the normal body weight of men of their heights and ages. The observed values averaged 17.83 liters, and the calculated normal mean was 16.33 liters.

After limited refeeding there was no significant change in the absolute plasma volume. The absolute thiocyanate space, however, tended to diminish somewhat during this refeeding—that is, the values came closer to those calculated for normal men of the same ages and heights. The relative volume of the thio-

TABLE 395

PLASMA VOLUMES, in liters per person and in cc. per kg. of body weight, in semi-starvation. Body weights, expressed as percentages of the normal average, are given under the heading "% Wt." The "normal" value was calculated for the normal weight after Henschel *et al.* (1947). "Excess" refers to the difference between the normal average of 45.3 cc. per kg. of body weight and that actually found. For each individual the initial data refer to the condition before refeeding. (Beattie, Herbert, and Bell, 1948.)

Subject	Date	% Wt.	Plasma Volume (liters)			Vol. cc./Kg. Wt.	
			Normal	Found	N. — F.	Found	Excess
Dutch							
v.A.	20/5	60	3.37	2.73	0.64	61.1	15.8
v.A.	11/6	62	3.37	2.91	0.46	63.0	17.7
d.G.	21/5	71	3.17	3.13	0.04	62.6	17.3
d.G.	12/6	79	3.17	3.63	—0.46	66.0	20.7
Z.	14/6	64	3.04	2.92	0.12	68.2	22.9
E.	15/6	73	3.44	3.84	—0.40	69.4	24.1
E.	28/6	75	3.44	3.08	0.36	73.5	28.2
J.	14/6	71	2.54	2.97	—0.43	74.0	28.7
J.	28/6	75	2.54	3.08	—0.54	73.5	28.2
V.	19/5	62	2.94	2.52	0.42	63.0	17.7
V.V.	21/5	67	2.83	2.67	0.16	63.4	18.1
V.V.	11/6	71	2.83	3.00	—0.17	68.0	22.7
T.	22/5	63	2.72	2.99	—0.27	78.7	33.4
T.	12/6	63	2.72	2.84	—0.12	74.7	29.4
K.	22/5	60	2.99	3.30	—0.31	82.9	37.6
Fr.	27/5	59	2.76	2.58	0.18	72.3	27.0
Fr.	13/6	61	2.76	2.50	0.26	67.2	21.9
D.	27/5	51	3.13	2.73	0.40	78.0	32.7
D.	13/6	56	3.13	3.30	—0.17	85.7	40.4
Vr.	1/6	53	2.65	2.29	0.36	74.4	29.1
Vr.	26/6	64	2.65	2.47	0.18	65.5	20.2
Initial M		62	2.97	2.89	0.08	70.6	25.4
German							
Ks.	31/7	78	3.22	3.51	—0.29	63.3	18.0
Ks.	25/9	81	3.22	3.39	—0.17	58.8	13.5
Lv.	30/7	75	3.31	3.49	—0.18	63.5	18.2
Kl.	30/7	82	2.94	3.06	—0.12	57.5	12.2
Kl.	23/9	85	2.94	2.79	0.15	50.5	5.2
Wz.	31/7	75	3.24	3.15	0.09	58.5	13.2
Sc.	30/7	81	3.13	3.34	—0.21	60.0	14.7
Sc.	24/9	85	3.13	3.14	—0.01	53.7	8.4
Gm.	1/8	71	3.19	3.25	—0.06	64.8	19.5
Gm.	15/9	73	3.19	3.00	0.19	58.0	12.7
Gt.	1/8	67	3.31	3.33	—0.02	70.5	25.2
Gt.	31/8	69	3.31	2.98	0.33	59.5	14.2
Rs.	1/8	83	3.04	3.10	—0.06	56.1	10.8
Rs.	25/9	83	3.04	2.88	0.16	51.5	6.2
Gd.	2/8	66	3.13	3.17	—0.04	69.7	24.4
Gd.	10/8	66	3.13	3.27	—0.14	72.2	26.9
Bm.	2/8	74	2.94	3.48	—0.54	66.9	21.6
Bm.	25/9	80	2.94	2.88	0.06	55.4	10.1
Ru.	2/8	77	3.17	2.99	0.18	55.7	10.4
Ru.	10/8	76	3.17	2.80	0.37	52.8	7.5
Initial M		75	3.15	3.26	—0.11	62.4	17.1

TABLE 396

THIOCYANATE (SCN) SPACE AND EDEMA IN SEMI-STARVED GERMAN CIVILIAN PRISONERS. "Normal" SCN space, in liters, was calculated as 23.5 per cent of the normal weight for height and age. (Beattie, Herbert, and Bell, 1948).

Subject and Date	SCN Space			SCN Edema		Clinical Edema
	Normal	Found	% of Wt.	Liters	% of Wt.	
Ke.						
31/7	16.69	18.98	34.2	5.94	10.7	+
25/9	16.69	18.09	31.3	4.46	7.7	0
Lv.						
30/7	17.16	18.76	34.1	5.83	10.6	+
Kl.						
30/7	15.28	16.76	31.5	4.26	8.0	0
23/9	15.28	16.45	29.6	3.38	6.1	0
Wz.						
31/7	16.80	18.61	34.6	5.97	11.1	+
Sc.						
30/7	16.22	19.33	34.7	6.24	11.2	+
24/7	16.22	18.91	32.3	5.16	8.8	0
Gm.						
1/8	16.57	17.49	34.9	5.72	11.4	+
15/9	16.57	16.93	29.2	3.32	5.7	0
Gt.						
1/8	17.16	18.02	36.9	6.53	13.4	+
31/9	17.16	16.06	32.1	4.29	8.6	0
Rs.						
31/7	15.75	18.03	32.6	5.03	9.1	0
25/9	15.75	16.96	30.2	3.76	6.7	0
Cd.						
2/8	16.22	16.01	35.2	5.32	11.7	+
10/8	16.22	15.34	33.9	4.71	10.4	0
Bm.						
30/7	15.28	15.52	32.4	4.26	8.9	0
24/9	15.28	15.55	29.7	3.25	6.2	0
Ru.						
2/8	16.45	18.66	34.8	6.06	11.3	+
10/8	16.45	18.20	34.3	5.73	10.8	0

cyanate space remained above normal in spite of the disappearance of clinical edema in this group. After 56 days of refeeding the mean thiocyanate space in 5 men was 30.6 per cent (range 29.6 to 32.3) of the actual body weight at that time. It must be noted that the level of refeeding used was sharply limited, rising by steps from a semi-starvation level of 1750 Cal. daily to a final maximum of 2530 Cal. daily for 13 days.

All these findings are in remarkable agreement with those obtained in the Minnesota Experiment, and the results of both series of studies substantiate the conclusion drawn by Beattie, Herbert, and Bell, as well as by the present authors, that the edema seen in most cases of ordinary semi-starvation represents simply a persistence in the body of its normal complement of extracellular fluid. Beattie and his group proposed the term "isohydric edema" for this condition. With more severe hypoproteinemia, however, the additional factor of low colloid osmotic pressure tends to produce a real expansion of the extracellular space and a more severe type of edema which may proceed to massive anasarca.

In these semi-starved persons Beattie and his colleagues observed that clinical edema could be perceived only when the thiocyanate space was in excess, compared with the normal proportion, by about 10 per cent of the body weight. The same value was estimated for this critical edema level by Henschel *et al.* (1947). If we translate these percentage values into absolute units, it means that an emaciated man of average height would have to contain about 6 liters of edema fluid before it could be recognized by ordinary clinical inspection. It is proper to record here that Dr. Beattie and the present authors discussed this matter, with mutual analyses of the pooled data relevant to famine edema, both in Minneapolis and in London. The close and detailed agreement between the findings of the Minnesota Experiment and those of the studies in the Netherlands and in Germany provides a very satisfactory basis for concluding that the results have general application.

Edema in Miners on the Island of Elba

In 1942 examination of about 500 laborers in the iron mines of the Island of Elba showed that 23 per cent of them had a moderate edema which was not explained by cardiac or renal studies. These men had been subsisting on a diet of about 1500 Cal., containing 78 gm. of protein, and exhibited the classic signs of caloric undernutrition — weight loss (10 to 15 or even 20 kg.), weakness and fatigue, sinus bradycardia, hypotension, and polyuria. Eight of these men were studied in detail while continuing to subsist on the low calorie diet and then were somewhat rehabilitated by administration of sugar and thiamine for 19 days.

In the edematous state these men had average values for the serum proteins: total 6.45 gm. per 100 cc. (range 5.46 to 7.89), albumin 4.59 (range 3.83 to 5.08). After treatment, and with the men free from edema, the corresponding values were: total 7.20 (range 5.44 to 7.97), albumin 5.33 (range 3.80 to 5.91). These results did not suggest any important role of hypoproteinemia or low colloid osmotic pressure to the Italian investigators.

These investigators (di Granati *et al.*, 1947) experimented with the addition of sucrose and of thiamine, singly and combined, to the dietary intake. Eight days with 200 gm. of sucrose daily produced no marked effect; 5 succeeding days with 25 mg. of thiamine daily did not affect the edema, but there was some increase in the pulse rate; 6 succeeding days with both the sucrose and the thiamine resulted in a disappearance of the edema and the bradycardia. Though the authors thought the thiamine was a valuable adjunct, particularly when the extra calories given these men were entirely in the form of sugar, the data do not indicate any previous thiamine deficiency. The 24-hour output of thiamine in the urine before treatment averaged 274 (range 104 to 428) micrograms. Moreover, the blood lactate level was not changed by the treatment except for a questionable *increase*.

Miscellaneous Observations during and after World War II

In spite of improved methods and knowledge and, unhappily, an abundance of famine victims, the measurements and studies on famine edema during and after World War II are few and generally disappointing. The Germans took no

advantage in this respect of the opportunities they were creating wholesale. The efforts of the Allied army nutritional teams were directed toward other ends, and the basic demands for relief precluded anything but hasty and superficial evaluations. The more significant studies, such as those of Beattie, Herbert, and Bell, of Govaerts, and of the Oxford Survey, have been discussed above. There are some other studies which merit briefer mention.

Heilmeyer (1946) reported blood studies on 4 patients with hunger edema, in 3 of whom the plasma C.O.P. was definitely low, but in one man this value (295 mm. of water) is reasonably close to normal averages. Heilmeyer added the observation that at least 90 per cent of the cases of hunger edema were seen in men; women only rarely were so affected.

The observations and clinical experiments of Petrides (1948) on nutritional edema in infants are of much interest in demonstrating the details of fluid shifts in purely hypoproteinemic edema. The finding that a high caloric but low protein intake will not suffice to remove the edema if the plasma proteins are severely depleted is not surprising. The results do not prove, as the author suggests, that all famine edema is simply a result of protein inadequacy.

The enormous difficulties of making precise observations in the chaos of war are evident in the reports of various workers. The wonder is not that the data obtained are often inadequate, but that any data at all could be collected. Some of these bits of evidence are useful in extending the broad picture of famine edema. For example, it is of interest that the total serum protein in 18 severely undernourished German prisoners of war in Austria averaged 5.7 gm. per 100 cc. (Davidson *et al.*, 1947). We may note also that Gsell (1945), who worked with patients from German concentration camps, concluded that hypoproteinemia is an obligatory condition for hunger edema and that the albumin fraction is most important. Gsell's discussion of the problem is descriptive and impressionistic, but he did supply details of the electrophoretic analysis of blood serums in 2 cases which he apparently considered typical (see Table 397).

Repatriated American prisoners of war from the Far East were examined by Wright and Van Ravenswaay in 1945 (Morgan, Wright, and Van Ravenswaay, 1946). Plasma proteins were measured in 1190 of these men, of whom 72 were edematous. The range for total plasma proteins was from 6.0 to 8.4 gm. per 100 cc., and only 77 men, of whom 16 had edema, had values of less than 6.5 gm. Of the entire group of 4618 men seen, edema was present in 10 per cent (465 men). At the time of examination a minority of these men exhibited vitamin deficiencies but 50 to 75 per cent of them had a history of wet or dry beriberi.

TABLE 397
SERUM PROTEINS, BY TISELIUS ANALYSIS (ELECTROPHORESIS), IN 2
PATIENTS WITH FAMINE EDEMA (Gsell, 1945).

Patient	Total Protein (gm./100 cc.)	Albu- min (%)	Globulin (%)			A/G
			% α	% β	% γ	
H	5.33	54.2	9.8	11.9	24.1	1.18
K	5.35	49.2	7.9	17.9	25.0	0.97

Leyton's (1946) valuable report on prisoners of war in a German prison camp contains no data on C.O.P. or A/G ratio, but the total plasma protein values for the British and Russian prisoners are of interest. None of the 61 British soldiers showed edema and only 5 of these men had plasma protein concentrations of less than 6 per cent. Among the Russian soldiers edema was present in all men where the plasma proteins were 4.7 gm. or less per 100 cc., and in a few cases at higher levels. The highest total plasma protein concentration at which edema was seen was 5.2 gm. per 100 cc. This series differs from all others in the fact that hypoproteinemia would seem to be sufficient to account for all the edema cases seen.

The most extreme conditions of starvation with all degrees of edema were found in the Belsen concentration camp. Random samples of the inmates gave an average of 5.1 gm. of protein per 100 cc. of serum; British soldiers on the spot gave a control average of 6.7 gm. (Mollison, 1946). In general, there was a tendency for the degree of edema to be related to the level of the plasma proteins in the Belsen inmates, but there were marked exceptions in numerous individuals. The results of these examinations conform to the general experience that the level of the plasma proteins affects the tendency to edema but, in famine at least, is by no means the controlling factor.

Edema and Diarrhea

In many, if not most, of the reports on famine edema it is striking to note the association with diarrhea. Generally, the sequence seems to be diarrhea and then edema. In recurrent cases the development of the cycle is repeated over and over again: diarrhea, edema, remission of both, and then diarrhea followed by edema. In the terminal stage of starvation the diarrhea may become continuous, edema subsides, and death occurs with the patient in a dehydrated state. This phenomenon is frequent, but in many other cases the patient is edematous at death.

Whether there is any direct causal relationship between diarrhea and edema is not clear. Certainly edema can develop with no sign of diarrhea at any time; this was the case in the Minnesota Experiment. But diarrhea often seems to predispose toward edema, even when extreme undernutrition is not involved. Berkeley (1948) reported 35 cases of edema in a series of 176 infants with gastroenteritis and diarrhea. In these infants, and in other cases of similar nature reported in the literature, Berkeley was unable to find any explanation from consideration of the plasma proteins, or of the heart.

Plasma Proteins and Colloid Osmotic Pressure in the Minnesota Experiment

The results of plasma protein analyses in the Minnesota Experiment have been discussed in Chapters 20 and 23. For consideration of the edema problem specifically, it is desirable to consider these proteins in terms of their colloid osmotic pressure. Equation (5) has been used for these calculations since it has proved entirely reliable in comparison with actual C.O.P. measurements in many hundreds of cases, including a large series of edematous patients. Further justification in the present work was found in the normality of the electrophoretic patterns in the serums from the Minnesota Experiment.

TABLE 398

MEAN COLLOID OSMOTIC PRESSURES calculated according to the method of Keys (1938) for 1° C. The values are in mm. of water for the 8-man groups fed at different caloric levels in rehabilitation. S24 = 24 weeks of semi-starvation; R12 = 12 weeks of rehabilitation.
(Minnesota Experiment.)

Group	Control	S24	R12
Z (basal diet)	294.1	255.9	296.6
L (+400 Cal.)	294.1	238.8	269.1
G (+800 Cal.)	291.5	256.6	261.2
T (+1200 Cal.)	303.8	256.6	304.5
M	295.9	252.0	283.3
SD	27.5	31.5	35.7

The summarized colloid osmotic pressure values are given in Table 398. The grand mean for 32 men in the control period was 295.9 mm. of water; this corresponds to about 315 mm. of water at 25° C. and is close to the averages for other control groups as noted above. The differences between the means for the 8-man groups in the control period were not great, ranging only from 292 to 304 mm. After 24 weeks of semi-starvation the mean colloid osmotic pressure declined in all 4 groups (from 34.9 to 55.3 mm.). Twelve weeks of rehabilitation produced increases in the C.O.P. in all groups, but the recovery at R12 was not related to the level of refeeding. For the entire group of 32 men the average at R12 was still below the control value although clinical edema was no longer present.

The Magnitude of the Edema in Semi-Starvation

One of the difficulties in discussing edema is the lack of a quantitative measure of the amount of edema present. When estimations of the extracellular fluid or thiocyanate space are available, it is possible to represent the edema as the excess of the extracellular fluid as compared with the normal proportion of this compartment of the body. As measured in the Laboratory of Physiological Hygiene the average for the thiocyanate space is about 23.5 per cent of the body weight in normal young men, and individual variations are not very great; in general, the extremes are 20 and 26 per cent. Thiocyanate space much in excess of this value represents edema, and the amount of edema may be expressed roughly as the amount of the excess.

We have calculated this excess in the Minnesota Experiment. At the end of semi-starvation (S24) the average excess thiocyanate space was 10.48 per cent of the body weight. The thiocyanate space represented an excessive proportion of the body in every man at this time, although not all of them exhibited clinical edema. At R12 there was, on the average, an excessive thiocyanate space amounting to 7.03 per cent of the body weight, but several men no longer showed any excess in the thiocyanate space.

In Figure 135 the relationship between the plasma colloid osmotic pressure and the magnitude of the edema, expressed as excess of thiocyanate space, is shown for observations at S24 and at R12. At the period of greatest edema, S24,

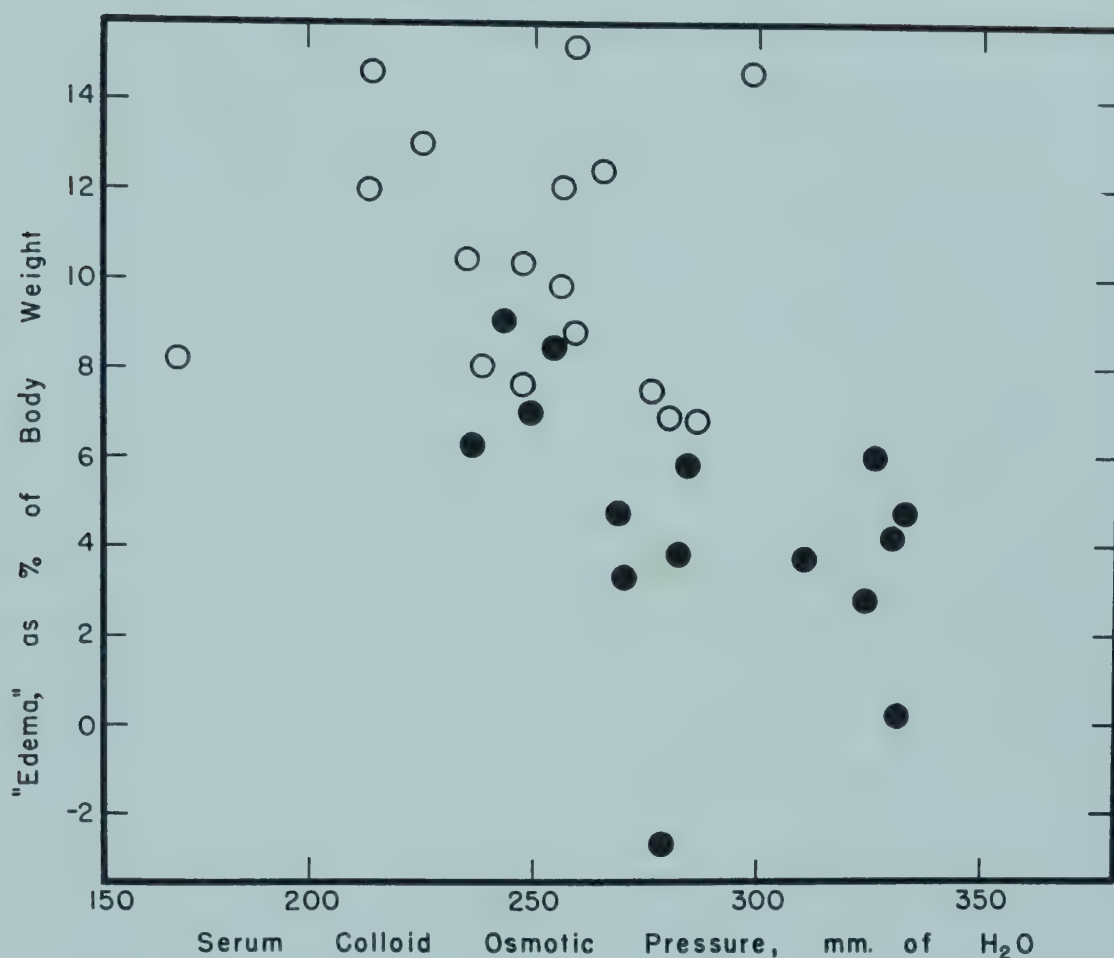


FIGURE 135. RELATIONSHIP BETWEEN CALCULATED SERUM COLLOID OSMOTIC PRESSURE AND "EDEMA," expressed as the excess percentage of the body weight represented by thiocyanate space. Open circles represent observations at 24 weeks of semi-starvation and solid circles at 12 weeks of rehabilitation. (Minnesota Experiment.)

the correlation was insignificant ($r = -0.118$), but a better correlation ($r = -0.432$) was found at R12. For the entire material there was a significant but not close inverse correlation between the "edema" and the C.O.P. ($r = -0.536$). These results are in agreement with all other information; the colloid osmotic pressure of the plasma affects, but does not control, edema formation in under-nutrition.

The actual amount of edema, estimated as excess thiocyanate space, was considerable in these men, ranging from about 4 to 8 or 9 kg. in the different subjects at S24. The largest individual edema accumulation was 15.2 per cent of the body weight. Clinically, 4 of the 32 men showed no palpable edema at any time and only 2 men were ever graded as 3+ on a scale of zero to 4+. This illustrates again the often demonstrated but frequently ignored fact that a very sizable amount of excess fluid must accumulate to be clinically recognizable.

The relation between the clinically estimated magnitude of the edema and the excess thiocyanate space is of interest (see Table 399). At the end of semi-starvation the edema was graded clinically on a scale of zero to 4+ by Dr. Rus-

TABLE 399

RELATIONSHIP BETWEEN CLINICAL EDEMA AND THE MAGNITUDE OF THE EDEMA ESTIMATED FROM THE THIOCYANATE SPACE. For the latter, it is estimated that in the normal, edema-free condition the thiocyanate space averages 23.5 per cent of the body weight. Here the tabulated figures are for the percentage excess of thiocyanate space over 23.5 per cent of the body weight. *N* = the number of men involved. (Data from the Minnesota Experiment at 24 weeks of semi-starvation.)

	<i>N</i>	<i>M</i>	Range
Normal	6	7.8	6.7-8.0
Edema 1+	7	10.9	7.6-15.2
Edema 2+ to 3+	5	12.2	8.2-14.6

sell M. Wilder in consultation with members of the Staff of the Laboratory of Physiological Hygiene. At about the same time the thiocyanate space was estimated in 18 of these men. The excess thiocyanate space—that is, the excess above 23.5 per cent of the body weight—averaged 7.8 per cent of the body weight in 6 of the men judged to be free from edema at that time. In 7 of the men graded as 1+ edema, the average was 10.9 per cent. In 5 of the men graded as 2+ to 3+ edema, the excess thiocyanate space average was 12.2 per cent of the body weight.

In general, the clinical estimate paralleled the excess of thiocyanate space but there was much overlapping. Perhaps the most interesting finding was that the excess thiocyanate space had to approximate 10 per cent of the body weight before it was clinically recognizable as edema. The “occult” edema amounted to about 4 kg., or 9 lbs., on the average in this small group (average weight 117 lbs. at the time). The addition of about 2.3 kg. (5.1 lbs.) of fluid was apparently enough, on the average, to convert a person considered clinically edema-free to one who would be graded 2+ to 3+.

Several of the Minnesota men developed rather large accumulations of fluid in the knee joints, enough to be troublesome while walking. Several checks on this point with all the men revealed a “floating” patella in 16 of them (that is, in 50 per cent of the group). Excluding the 4 men who never showed any clinical edema, the incidence of floating patella amounted to 57 per cent. This seems to us a large percentage in view of the generally rather moderate extent of the edema in these men.

Repeated attempts were made to discover the presence of ascites or pleural fluid in these men, with entirely negative results. This is in accord with the relative rarity of ascites and hydrothorax observed in most famine victims, except in the Orient.

There are a few other data on the extracellular fluid space, as estimated by the thiocyanate method, in famine edema. In 15 French patients there was a relative expansion of this space, but not necessarily any absolute abnormality (Cachera and Barbier, 1943), and much the same condition seems to have been found among German civilian internees (Denz, 1947). Denz emphasized the fact that the clinical estimation of edema “apparently gives no certain measure of the degree of hydration of the body” (p. 12). He pointed out that large

amounts of excess fluid may be present where, to external examination, the subcutaneous tissues reveal little edema. One of his patients, who had only slight edema of the ankles, lost 8.5 kg. in weight in a few days, although Denz hazarded the opinion that the visible edema "could at the most account for one litre."

Beattie and Herbert (1948) made important studies on the extracellular fluid space which are discussed in a separate section of this chapter; in general they are in very good agreement with the Minnesota findings. Kerpel-Fronius and Kovach (1948) have briefly reported results with the thiocyanate method on 23 infants, 10 of whom were from 30 to more than 50 per cent underweight; the cause of the underweight was simple semi-starvation in Budapest. The results showed that there were only slight differences in the absolute amounts of the thiocyanate space in all of these infants but that the percentage of the actual body weight which this represented was markedly affected by undernutrition. In the infants with body weights between 80 and 120 per cent of the ideal weight the thiocyanate space represented roughly 26 to 34 per cent of the actual weight, but in the infants weighing only 45 to 70 per cent of ideal weight the thiocyanate space amounted to from 40 to 50 per cent of the actual total body weight.

Explanation of the Edema in the Minnesota Experiment

In the Minnesota Experiment at least, the explanation of the edema must be left largely in negative terms. Effective hypoproteinemia or lowered colloid osmotic pressure, increased capillary permeability to colloids or excessive proteins in the edema fluid, elevated venous or capillary blood pressure—none of these can be seriously entertained here. The trivial decline in the plasma proteins can be offset against the much more substantial decline in the venous blood pressure. Renal insufficiency, even if it did exist, and there is not the slightest evidence that it did, could not produce the actual mechanism to cause excessive transudation. And, as far as the evidence goes, the situation in the Minnesota Experiment was much the same as in famine edema seen in the field in many instances; in other words the same difficulty in finding an explanation for the edema occurs in the field and in the laboratory.

Obviously, the whole picture would be different if it could be maintained that data on the plasma proteins, and the A/G ratio obtained by ordinary standard methods, are inadequate for even a rough characterization of the colloid osmotic pressure. Practically none of the studies to date on the blood in starvation would then have any necessary relevance to the edema problem. But such an extreme position cannot be defended and the interesting findings of Govaerts and of Lamy and his colleagues merely suggest that some small part of the plasma proteins becomes less than usually effective, osmotically, in starvation. Most of the problem remains unsolved.

In the face of the foregoing and in the absence of other information, further argument is necessarily conjectural. Perhaps the hydrostatic gradient in the arterioles and capillaries was abnormal. A relative relaxation of the arterioles and a relative constriction of the venous end of the capillaries might conceivably allow a greater net filtering pressure in the capillary and still allow an

abnormally low venous pressure to exist. It is doubtful whether such conditions could be produced. An impediment to lymphatic return might allow an accumulation of fluid in the tissue spaces, where, undoubtedly, there was a real reduction in elastic pressure. It may be that elastic tissue pressure external to the capillary is normally much higher than is commonly thought to be the case; if this were so then there might be room for an effective decline in this pressure in starvation.

It will have been noted in Table 152 that the thiocyanate space stayed curiously constant in absolute amount; does this have any special meaning in relation to elastic forces or tissue structure? The edematous outcome could be viewed as merely the result of a shrinkage of the cellular components without much change in the absolute amount of interstitial fluid or of the blood volume. The question could be asked, perhaps naïvely, as to why the extracellular fluid should adjust itself to the diminished cellular mass of the body. Moreover, what forces exist to bring about such adjustment? The kidney, as we have seen, is entirely competent and satisfactorily performs its function of regulating the concentrations of water, salts, and urea in the blood. There is polyuria because the starving man drinks more water and eats more salt. Unless we presume the operation of some unknown factors, it is hard to see why and how the interstitial fluid and blood volume should change, in absolute amounts, in starvation.

Renal function in cases of famine edema requires much further study. It may be, as Denz (1947) suggests, that systematic and detailed research on glomerular and tubular function would clarify matters. It is uncertain, however, just how it would be possible for the kidney to control edema formation and subsidence without acting by way of the several pressures at the general tissue capillaries.

It must be abundantly clear that the balance of fluid between blood and tissue spaces is not a simple equilibrium and that the Starling equation is an oversimplification of a dynamic steady state. The attempt to treat this state by equilibrium mass considerations is not justifiable in terms of physical chemistry. Fluid is constantly moving in at least three ways — out of the blood into the tissue spaces, back from the tissue spaces into the blood, and away from this locus by the lymphatic channels. The resultant, which is the amount of fluid in the tissue spaces, depends upon the rates of these several movements. And it is well known that rates cannot be predicted from equilibrium equations.

Mixed Edemas

It should be obvious from the foregoing discussion that a number of factors may, and often do, contribute to the edema seen in calorically undernourished persons. In "natural" series of such cases it may be expected that some patients will exhibit sufficient hypoproteinemia to account, at least superficially, for the edema. It is improper to conclude that famine edema is produced by hypoproteinemia simply because the *average* protein level is rather low in such patients or that some individuals with marked edema also have very low plasma protein levels; this is a logical error which is all too common (cf. Govaerts, 1947; Kurnick, 1948). It would be equally unjustifiable to deny an effect of the plasma proteins because many cases do not show a significant hypoproteinemia or hypoalbuminemia. The emaciated individual may have edema with normal plasma

proteins but, other things being equal, he should have more edema if he is hypoproteinemic. In the same way, cardiac failure will increase the edema tendency.

In the Minnesota Experiment, where other complications were at a minimum, the edema in all cases seems to have been independent of the level of plasma proteins. In the Dutch and German subjects studied by Beattie, Herbert, and Bell (1948) this was the case with all but a very few individuals such as their patient F., who had massive edema and a total serum protein level of 4.60 gm. per 100 cc. In the Saipan population studied by Kurnick (1948) some 78 per cent of the edematous patients had serum protein levels below 5.5 gm. per 100 cc., and about 26 per cent of them had values of 3.5 gm. per 100 cc. or less. In most of Kurnick's patients, then, it is certain that hypoproteinemia contributed to the picture, but one is entitled to ask about the 17 per cent of the patients with serum protein concentrations of 6.1 gm. per 100 cc. and higher.

With such data as those of Kurnick the usual argument is that, had the albumin been separately determined, the explanation might be forthcoming. But this is specious, because when albumins are reported there is rarely much change in the number of cases unexplained by the simple colloid osmotic theory. This is the case in simple famine; in nephrosis, in some forms of liver disease, and probably in "war nephritis," the predilection for a differential albuminemia is greater. By and large, it would seem that whenever there is extreme edema it is certain that factors other than caloric deficiency are operating. If the patient is also severely undernourished calorically, the edema is of the mixed type.

Edema in Acute Starvation (Fasting)

The many differences between acute (total) starvation and semi-starvation have been emphasized repeatedly in this book. One notable point is the absence of the development of clinical edema in fasting. But, as already indicated, large changes in extracellular fluid may take place without detection by clinical inspection. What actually happens to the size of the fluid compartments of the body in prolonged fasting?

A recent study on one man, who lost about 30 per cent of his original body weight in 45 days of fasting, provides interesting information (Sunderman, 1947). This man was examined at the end of his fast and again after 43 days of refeeding when he had regained most of his weight loss. There was no clinical edema at any time. The essential data are given in Table 400. In the starved state the plasma volume and the thiocyanate space were both markedly elevated

TABLE 400

ACUTE STARVATION DATA ON ONE SUBJECT, at the end of a 45-day fast and after 43 days of subsequent refeeding. The total base is given in milliequivalents per liter, the protein values in gm. per 100 cc. (Values from Sunderman, 1947.)

	Body Weight (lbs.)	Serum Concentration				Serum Volume		SCN Space	
		Total Base	Total Proteins	Albu- min	Globu- lin	Total (liters)	Cc./ Kg. Wt.	Total (liters)	% Body Wt.
After fasting	97	141	6.2	5.0	1.2	2.54	57.5	14.7	33.4
After refeeding	134	148	6.7	4.0	2.7	2.88	47.0	14.6	24.0

when calculated per unit of the actual body weight of the subject at that time; his body was overly hydrated, in these compartments of the body at least. However, if these fluid volumes are computed on the basis of the subject's previous weight, or the normal weight for his height, they are well within normal limits. With refeeding, there was a slight increase in absolute plasma volume and no change in the absolute thiocyanate space.

These findings in total starvation are in complete agreement with those of the Minnesota Experiment on semi-starvation, and Sunderman's data find their counterpart in those from some of the Minnesota men who were free from clinical edema at the time of the measurement of the thiocyanate space. From the Minnesota evidence a value of 33.4 per cent of the body weight in the extracellular space is, in an emaciated man, at the level where clinical edema may or may not be recognizable. One might predict that if Sunderman's subject could have been induced to drink a little salt water during fasting he would have shown clinical edema. Finally, it may be noted that in this fasting subject the analyses of the serum proteins provide no possible explanation for the development or subsidence of the relative hydremia and excessive proportion of the body occupied by extracellular fluid.

Aberrant Edemas Associated with Undernutrition

In times of famine the great majority of edema cases correspond to a classical picture: dependent edema associated with weakness, bradycardia, polyuria, depression, moderate anemia, slight to moderate reduction in plasma protein concentration, and emaciation. But cases which clearly depart from this picture occur from time to time. In general, the detailed nutritional picture in these aberrant cases is not clear, nor has the possibility of concurrent infections and other abnormalities been properly explored. Individual idiosyncrasies do not merit discussion here, but in some instances there were considerable numbers of individuals who showed similar peculiarities. Some of these are of interest even though in none of them are there adequate explanations for the unusual findings.

The long history of famine and edema in India contains many references to "epidemic dropsy," which, in some instances at least, clearly departed in character from the more ordinary type of famine edema seen in both Europe and Asia (cf. Leys, 1920). Greig (1912) reported many cases of rapidly developing edema associated with food shortages; these persons exhibited pyrexia, dyspnea, and vomiting, none of which is ordinarily seen in famine edema. In 630 patients, 322 were febrile, 364 had dyspnea, 106 complained of cardiac palpitation, 15 had disturbances of vision associated with increased intraocular tension, and 180 had mottling and petechia of the skin. All these patients were anemic and in none could there be found evidence of an infectious basis. Greig noted that the incidence and subsidence of this condition, as examined in a number of outbreaks in different years, coincided closely with the price of grain and other evidences of waxing and waning undernutrition.

Ray (1927) stated that the epidemic dropsy which occurred in Bengal in 1926 was the same as had occurred in India in previous outbreaks and that Norman Chevers had labeled it *morbus Bengalensis* in 1877. In any case, the edema was associated with undernutrition but not necessarily of the severest degree.

the fatalities were generally due to heart failure, and there were many signs of cardiac involvement. There was marked anemia. The total serum protein concentration was about normal, but the albumin fraction was generally much reduced. The general appearance of mottled erythema of the skin and the frequency of angiomatous growths were notable. Certainly one must continue to suspect some infectious or toxic basis for these "epidemic dropsies"; the coincident bad nutritional state would seem to be a complicating factor in the picture.

At least one form of epidemic dropsy in India seems to be a result of a toxic factor derived from mustard (argemone) oil (Mitra and Rao, 1947). The malady is of sudden onset, with abdominal complaints, dependent edema, general malaise, and arthralgia. Men aged 20 to 40 years complained of impotence. Confusion with famine edema is possible because mustard oil is consumed in India only in times of food shortage, but the clinical picture is quite distinct.

The "generalized edema" reported from Rangoon by Kundu (1934) is obviously another aberrant form quite distinct from the Bengal disease. The patients were not severely starved though they were certainly malnourished, rice making up by far the greater part of the diet. They had no signs of renal disease, beriberi, or parasitic infestation. There was moderate anemia, polyuria, and rapidly progressive edema and anasarca. Improvement was rapid on a good diet. The peculiarities in this Burmese edema were the relatively slight amount of real starvation involved and the great severity of the edema. An unusual degree of protein deficiency may be suspected.

One of the most curious aberrant edemas was reported from Haiti by Mann, Helm, and Brown (1920). Some 3000 cases and 200 necropsies were observed, chiefly in prison inmates, and local information was that the disease had been endemic on the island for more than 40 years. The rapidity of development, the high mortality, and the relative resistance to dietary treatment were outstanding points of difference in comparison with all the usual forms of famine edema. In one group of 40 bandits, all of whom were in good physical condition when imprisoned, there was one case of edema within 12 days and 10 cases within 7 weeks. However, the weakness, bradycardia, diarrhea, emaciation, and subnormal body temperature all resemble ordinary famine. The authors insisted that dietary measures had been "given a rather extensive trial, with results which were not at all gratifying" (p. 1418).

General Comment

It is apparent from the evidence summarized in this chapter that there is less disagreement about the facts of famine edema than in the conclusions which have been drawn from these facts. In this area of study, as in other fields of biology and medicine, it is only too easy to discover factors which are related to or which influence the phenomena in question and to conclude that these phenomena are thereby "explained." Such oversimplification delays the development of true understanding and is the source of endless and needless controversy. The relevant facts about which there seems to be little doubt may be summarized as follows:

1. The general character of the pressure relationships which affect fluid ex-

changes across the capillaries, including those in all forms of edema, are properly indicated in the Starling concept and in the general equation in which this is customarily expressed.

2. Neither physical-chemical theory nor empirical observation supports the view that the Starling equation can be rigorously applied to non-equilibrium states. Even where there is a steady state (no net tendency toward a change in the extravascular fluid volume) the Starling equation ignores the complications of lymph flow and the pericapillary circulation.

3. Except in complicated cases, the venous pressure in famine edema is subnormal, and it must be inferred that the intracapillary pressure corresponds. Excessive outward filtration as a result of pressure backing up from the heart cannot be entertained, but rather the indications are that there is a subnormal hydrostatic pressure in the direction of outward filtration.

4. Famine edema frequently develops in the presence of protein concentrations (total and albumin) which are well within normal limits. Hypoproteinemia, or hypoalbuminemia, or both, fosters the development of famine edema but is not essential to it. The development, magnitude, and disappearance of famine edema may be, and often are, relatively independent of changes in the concentrations of the plasma proteins.

5. The effective molecular sizes, and therefore the colloid osmotic pressure effects, of one or more proteins in the plasma may be somewhat altered in severe and prolonged undernutrition. If there are such changes, they involve only small proportions of the total proteins and produce only slight changes in the colloid osmotic pressure per unit of total protein concentration.

6. In famine edema the capillary walls ordinarily do not become unduly permeable to colloids and the colloid osmotic pressure of the tissue fluid is no greater, and probably is considerably less, than in normal non-edematous persons.

7. General hydrostatic pressure in the tissues is low in famine edema. No valid estimations are available for the actual hydrostatic pressure in the tissues immediately outside the capillaries.

8. The magnitude of the edema in famine edema can be altered somewhat by the intake of salt and water, but these items do not ordinarily control or closely determine the net fluid exchanges and balance.

9. Renal function is not grossly abnormal in famine edema. In any case, if the kidneys do exercise a prominent influence on famine edema, they must do so through mechanisms operating at or close to the capillaries.

10. The clinical recognition of edema, at least in famine edema, requires a very substantial elevation in the proportion of the body represented by extracellular fluid. In severe and prolonged undernutrition a relative increase in the extracellular fluid is almost always present, even though the edema is not clinically recognizable.

11. In simple famine edema uncomplicated by unusual anemia, hypoproteinemia, or intercurrent cardiac disease, the absolute volumes of both blood and extracellular fluids approximate the usual levels for normal persons of equal height and for edematous persons at their normal (pre-starvation) weights.

12. In such uncomplicated cases of famine edema it is reasonable to believe

that the tendency toward a small decrease in plasma colloid osmotic pressure and toward a probable small decline in tissue pressure is offset by a fall in intracapillary pressure, so that there is no significant resultant pressure to produce an important change in the net fluid exchange at the capillary. If this is so, the absolute volume of extravascular fluid would be substantially constant but the progressive loss of cellular bulk in the tissues would produce a relative increase in hydration of the body as a whole. The general tendency toward edema would be explained, then, without recourse to any theory about altered fluid exchanges at the capillary wall.

Anorexia Nervosa and Pituitary Cachexia

It is interesting to compare with both wartime starvation and famine, and the Minnesota Experiment, other types of semi-starvation in which living conditions, except the amount of food, most nearly approach normal. Anorexia nervosa and pituitary cachexia (Simmonds' disease, cachexia hypophyseopriva) are obviously conditions of great interest in this regard. To these conditions might be added Sheehan's disease (Sheehan, 1937; Sheehan and Murdoch, 1938), which involves necrosis of the pituitary and therefore has something in common with Simmonds' disease. To some extent they may be considered together for the present purpose.

Anorexia nervosa is a loss of the desire for food in the absence, in general, of significant organic disease; the food restriction is entirely psychological and is directly related neither to economic status nor to the availability of food. Almost all cases are women. There are frequently associated psychic disturbances which may profoundly alter the effective environmental situation, but generally these patients are not subject to the adverse living conditions that so complicate the situation in famine. Many excellent reviews of anorexia nervosa have been written, but these naturally emphasize the clinical problems of diagnosis and management (Dejerine and Gauckler, 1913; Berkman, 1930, 1948; Ryle, 1936; McCullagh and Tupper, 1940; Oppenheimer, 1944; Berkman, Weir, and Kepler, 1947).

Simmonds' disease, or hypophyseal cachexia (cachexia hypophyseopriva), is a cachectic state associated with severe pathological interference with the function of the adenohypophysis. Though the condition is increasingly diagnosed in life and occasionally such diagnoses are verified at autopsy, the fact remains that a differentiation from anorexia nervosa is often considered to be difficult. For our present purpose it is enough to note that patients with Simmonds' disease often maintain normal living conditions except for food intake until they present themselves in an emaciated state to the physicians who make the diagnosis. With certain exceptions which will be noted later, typical cases of Simmonds' disease are relatively free from associated diseases and abnormalities. Like anorexia nervosa, Simmonds' disease has much appeal to medical writers and the literature is voluminous. Escamilla and Lissner (1943) collected 101 proved cases out of 595 diagnosed cases in a literature of some 500 published reports.

It is not the purpose here to examine all or even an appreciable part of this material, but rather to discuss those aspects of anorexia nervosa and Simmonds'

disease which demonstrate the universality of some effects of caloric undernutrition, as well as to point out some features that are peculiar when compared with the effects of famine and enforced starvation.

Berkman (1948) has pointed out that in the emaciated patient with anorexia nervosa there is anterior pituitary insufficiency as a result of starvation and that differentiation from anterior pituitary insufficiency may be impossible on the basis of symptoms, signs, and laboratory findings, though with consideration of the history and behavior "the distinction usually can be made with ease" (p. 240). From the experience at the Mayo Clinic it is concluded that "a severe degree of cachexia is a very rare occurrence in a patient proved to have anterior pituitary insufficiency due to organic disease. Conversely, a patient who is extremely emaciated, rarely is found to have organic disease of the pituitary body" (p. 241).

Weight Losses in Anorexia Nervosa

The weight loss in anorexia nervosa patients is comparable to anything seen in famines, and probably greater nonfatal cachexia can occur in anorexia nervosa than in any other condition. There are abundant reports of the loss of more than one third of the body weight; an extreme but not unique case is one reported by Clow (1932): a woman reduced from 62.6 to 24.0 kg. (138 to 53 lbs.). Berkman,

TABLE 401
ANOREXIA NERVOSA. Body and organ weights at death in 2 female patients: Case A (from L. Stephens, 1895) and Case B (from Oppenheimer, 1944).

	Case A	Case B
Age at death (yrs.)	16	61
Body length (cm.)	162	152
Body weight (kg.)	22.2	29.9
Organ weight, in gm.		
Heart	113	150
Brain	1290	
Liver	675	850
Spleen	35	95
Kidneys	156	

TABLE 402
ANOREXIA NERVOSA PATIENTS WITH EXTREME WEIGHT LOSS AND SUBSEQUENT RECOVERY. Cases 1 and 2 are from Bruckner, Wies, and Lavietes (1938); Case 7 is from Berkman, Weir, and Kepler (1947).

Case	Age (yrs.)	Height (cm.)	Weight (kg.)			
			Low	Normal	% Normal	Recovery
1.....	16	141.0	22.0	46.3	47.5	40.8
2.....	18	152.4	24.5	50.8	48.2	55.1
7.....	32	158.8	27.0	57.6	46.9	36.3*

* After 82 days of treatment; great improvement continuing.

Weir, and Kepler (1947) observed a woman aged 24 years who was 5'2" (157.5 cm.) tall and weighed only 50 lbs. (22.7 kg.) but who had weighed 122 lbs. (55.3 kg.) 18 months previously. The standard weight for this age and height is 121 lbs. (54.9 kg.), so she was 58.7 per cent underweight and had lost 59.0 per cent of her weight. The authors stated (p. 364): "The patient was able to be up and about and was reasonably active. The value for serum protein was 7.2 gm. per 100 cc. She was not anemic. This patient did not remain for treatment."

The degree of wasting is well shown in the case reported by L. Stephens (1895). The patient was a 16-year-old girl who had been entirely normal until 10 months before death; at autopsy (56 hours after death) the body measured 162.6 cm. (5'4") in length and weighed 22.2 kg. (49 lbs.). Oppenheimer (1944) reported a case of a woman who died after a 27-year history of anorexia nervosa; her weight not long before death was 29.9 kg. (66 lbs.) and the body length was 152.3 cm. (59½"). The organ weights in these cases are summarized in Table 401. Weight losses of the body organs correspond to the body weight loss in much the same way as in starvation from other causes; as in other conditions of emaciation the brain weight changes little.

Although there is much variation, a weight loss of 50 per cent, or a reduction to 50 per cent of the normal average for height, has been suggested as the critical level in determining recovery (Dejerine and Gauckler, 1913; McCullagh and Tupper, 1940). But recovery from this degree of emaciation does occur in some cases, as indicated in Table 402.

Similarity between Famine Victims and Patients with Anorexia Nervosa

At equal degrees of weight loss, anorexia nervosa patients are similar to famine victims in numerous respects: the pulse is slow, averaging about 50; the blood pressure is low, rarely above 120 mm. Hg. systolic; amenorrhea is almost universal. The basal metabolic rate is reduced to an average of -30 per cent (Escamilla and Lissner, 1942), but values down to -73 per cent have been obtained (Sheldon, 1937). The basal metabolic rate in anorexia nervosa frequently lags behind the weight gain during convalescence (Farquharson, 1938, 1941). The body temperature is low; in two patients who later recovered, Bruckner, Wies, and Laviates (1938) recorded rectal temperatures of 95.2 and 96.0° F. (35.1 and 35.6° C.). Significant biochemical findings include normal to low cholesterol concentration in the blood serum (Stephens, 1941; Pardee, 1941), not related to the extent of loss of weight, and relatively normal serum protein values except when there has been extreme weight loss (Stephens, 1941; Bruckner *et al.*, 1938). Moderate anemia is nearly always present and is usually hypochromic in nature (Bruckner *et al.*, 1938; McCullagh and Tupper, 1940; Stephens, 1941). Evans (1939) has reported an instance of severe anemia responding to iron without substantial increase in caloric intake.

Elsewhere in the present work we have discussed the peculiar pigmentation of the skin which develops in some persons in famine areas and was seen in some of our own subjects in the Minnesota Experiment. This phenomenon also occurs in anorexia nervosa and is observed in perhaps 10 per cent of the patients (Berkman, 1930).

Differences between Victims of Anorexia and of Famine

The anorexia nervosa patient and the famine victim differ in certain interesting respects. Since the two meet only on the common ground of caloric restriction, it would seem probable that the different effects of semi-starvation under famine conditions must be related in large part to other deleterious factors. However, it is still not possible to be sure that special endocrinological disturbances may not be involved in some cases of anorexia nervosa.

The mental state in anorexia nervosa is primarily, of course, a product of the fundamental personality disturbance, and evaluation of the effects of semi-starvation must take this into account as well as the status prior to weight loss. The only known invariable difference between the psychological characteristics of anorexia nervosa and famine is the latter's continual hunger and mental absorption with food. Although some anorexia nervosa patients are reticent, apathetic, and depressed like famine victims, a considerable number show a feverish press of activity (Ryle, 1936; Magendantz and Proger, 1940), at times manic in character, extending even into the most severe cachectic phase. A similar state of excitement in famine victims is very rare and short-lived in any case.

There are many excellent descriptions of the psychic state in anorexia nervosa (e.g. Brosin and Apfelbach, 1941), and it is to be emphasized here that the mental change occurring in famine (described elsewhere in this book especially for the Minnesota subjects) is not entirely a simple and direct result of caloric restriction. Semi-starvation produces physiological changes which, in general, tend to push the emotional state and personality characteristics in certain directions. However, there are marked individual differences in the degree of personality change and deterioration in famine victims as well as in anorexia nervosa patients.

Muscular weakness has been stated to be less pronounced in anorexia nervosa than in other types of severe undernutrition. Grading muscular weakness from 0 to 4 in 117 patients, Berkman (1930) found no weakness in 9 per cent and grades 3 and 4 weakness in only 21 per cent of these patients. The significance of such figures is limited, of course, in the absence of detailed estimates of the corresponding weight losses. There is no doubt that many patients with anorexia nervosa do not exhibit the behavior expected in patients with severe muscular weakness, but this may be related to the tendency to excitement. With the most severe weight losses the prostration of the anorexia nervosa patient is apparently entirely similar to that of the victims of chronic famine.

There seems to be a notable difference with regard to atrophy of the breasts between famine victims and patients with anorexia nervosa. In the semi-starvation of famine adult women commonly suffer atrophy of the breasts, and this may be extreme. In the Indian famine of 1877-78 atrophy of the breasts was so marked that the gland seemed almost to have disappeared (Porter, 1889; Sheldon, 1937). Similar reports from other periods of famine are plentiful. In contrast, in cachectic women with anorexia nervosa breast atrophy is frequently not severe, even at death from starvation. In the extreme emaciation of Stephens' (1895) patient, only the breasts were spared. A notably normal appearance of the breasts in emaciated patients with anorexia nervosa has often caused com-

ment (Marshall, 1895; Sheldon, 1937; Bruckner *et al.*, 1938). Berkman, Weir, and Kepler (1947) did not comment on this point, but the photographs in their paper show cases of remarkable preservation of breast tissue in the presence of extreme general emaciation.

Simmonds' Disease

Simmonds' disease may, in some cases, be accurately diagnosed before emaciation has developed and may be verified at autopsy in patients who die from causes other than cachexia. Here at least we are concerned only with comparisons between ordinary starvation and Simmonds' disease where the degree of cachexia or weight loss may be comparable. It is dangerous to use such excellent tabulations as those of Escamilla and Lissner (1942) without making allowances for these facts. In the 101 proved cases analyzed by Escamilla and Lissner, cachexia was marked in only 65 per cent, but "asthenia" was reported in 90 per cent; in only 32 per cent of the verified cases were the reported data adequate for making a calculation of the amount of weight lost. Exact comparison with ordinary semi-starvation is also difficult because of the usual faults so common to clinical reports — normal or negative findings are often not mentioned, the conditions of measurement are not described, and the normal standards used for comparison are not validated.

With the foregoing limitations in mind, some valuable comparisons are still possible. Definite bradycardia is at least fairly frequent in Simmonds' disease, but it is by no means the rule. The pulse rate was mentioned in only 42 of the verified cases found in the literature and in only 21 per cent of these was the pulse rate as low as 60 or less. Escamilla and Lissner remarked that since the basal metabolism "is so consistently low, it is surprising that bradycardia does not occur more frequently" (p. 77). The blood pressure tends to be low, although mild hypertension may be found. In some cases hypotension is so marked as to suggest Addison's disease, but the usual picture is a moderately low blood pressure quite similar to that in ordinary starvation. In other respects also the cachexia of Simmonds' disease seems not to be peculiar; mention may be made of the hemoglobin concentration, blood sugar, body temperature, blood morphology, the incidence of amenorrhea, and dryness of the skin.

Besides the relative infrequency of bradycardia, patients with Simmonds' disease seem to differ, on the average, from victims of simple starvation in the high percentage of cases with loss of hair of the eyebrows, scalp, and beard and with excessive decay or loss of teeth. The loss or absence of sexual hair (axillary and pubic hair) in patients with Simmonds' disease is striking. In the verified series of Escamilla and Lissner 80 per cent of the patients exhibited this abnormality. While some loss of hair, including sexual hair, does occur in some famine victims, it is rarely as severe as may be seen in some patients with Simmonds' disease. Famine victims, like patients with Simmonds' disease, have a loss of libido, but the effects on the genital organs do not seem to be comparable to those reported for many cases of Simmonds' disease. It is reasonable to ascribe all these differences in primary and secondary sexual disturbances in Simmonds' disease to the special endocrine pathology in this disease.

It may be that the special characteristics seen in patients with Simmonds' dis-

ease would be eliminated in part or even wholly by a more rigorous exclusion of patients with associated pathologies. But until this is done, and until more exact laboratory and functional studies are made on such patients, it would seem to be unsafe to use the patient with Simmonds' disease as an example of simple semi-starvation or, conversely, to suggest that the characteristics of simple semi-starvation may be used in detail to describe the patient with Simmonds' disease.

Vitamin Deficiencies in Anorexia Nervosa and Simmonds' Disease

Vitamin deficiency conditions, particularly scurvy, beriberi, and pellagra, are more or less prominent in many populations under famine conditions. They were not present in the Minnesota Experiment and are rare in anorexia nervosa (Ryle, 1936; Bruckner *et al.*, 1938; McCullagh and Tupper, 1940; Stephens, 1941). Spongy gums are sometimes mentioned, and at least one instance of real athiaminosis has been reported in anorexia nervosa (Palmer, 1939). Ryle (1936) described an anorexia nervosa patient who showed signs of scurvy and died of purpura hemorrhagica. Berkman (1930) found a case of pellagra in his series of anorexia nervosa. We may ignore the attribution of amenorrhea in anorexia nervosa to vitamin E deficiency (Spence, in Ryle *et al.*, 1939; McCullagh and Tupper, 1940).

We have been unable to find much useful information about vitamin deficiencies in Simmonds' disease. Moss (1942) reported a patient with diabetes insipidus and Simmonds' disease who exhibited signs of multiple vitamin deficiencies. In the absence of positive reports and any mention of vitamin administration in case reports, we can assume that frank vitamin deficiencies are rare in this condition.

It seems safe to conclude that neither in anorexia nervosa nor in Simmonds' disease are there apt to be vitamin deficiencies, though some of the characteristics of these conditions resemble symptoms that are sometimes supposed to be associated with vitamin deficiencies. Among these may be mentioned dry skin, anorexia, weakness, bradycardia, and depression. These facts add force to the belief that these characteristics are related to caloric undernutrition and only exceptionally to vitamin deficiencies.

The Cause of Death and Some Remarks on Infections

In the earliest discussions it was stated that few patients with anorexia nervosa actually die of starvation (Lasègue, 1873). This opinion has not been borne out as clinical experience has accumulated, although the mortality directly attributable to anorexia nervosa is only something like 8 per cent. The findings at autopsy usually reveal little evidence of infection, mild and apparently only terminal pyogenic infection being most common (Marshall, 1895; Stephens, 1895; Conybeare, 1930; Ryle, 1936; Richardson, 1939; McCullagh and Tupper, 1940). Although gastrointestinal complaints are universal in anorexia nervosa, gastrointestinal infections are seldom seen.

Escamilla and Lissner (1942) discovered only 18 cases of tuberculosis in their compilation of 101 verified cases of Simmonds' disease, and only 3 per cent of 158 typical but unverified cases had tuberculosis. The same authors found no

tuberculosis in their analysis of 20 cases of anorexia nervosa; however, these patients do develop tuberculosis occasionally (Ryle, 1936; Brosin and Apfelbach, 1941). But careful scrutiny of the literature on both anorexia nervosa and Simmonds' disease suggests that tuberculosis is far less frequently a complication than might be expected in view of the cachexia and therefore presumed susceptibility to tuberculosis. The difficulties of diagnosis in both anorexia nervosa and Simmonds' disease undoubtedly distort the true incidence of tuberculosis as a complication. Frequently the diagnosis is in part or even to a large extent made by excluding other causes for the cachexia. A cachectic person complaining chiefly of loss of weight is always suspected of tuberculosis, and if this disease is found the question often is considered settled and there is not likely to be further speculation about any coincidental causes for the cachexia. It seems proper to conclude that the true incidence and mortality of tuberculosis in both anorexia nervosa and Simmonds' disease are unknown.

In the case reports on anorexia nervosa there is little or no mention of increased susceptibility to pneumonia, upper respiratory infections, rheumatic fever, or serious pyogenic infections. Much the same is true of the reports on Simmonds' disease. From the evidence in these two conditions it would be difficult to support the thesis that undernourishment leads to an important reduction in resistance to infection.

Edema in Anorexia Nervosa and Simmonds' Disease

Edema is fairly common in war famine and appeared promptly in most of the subjects in the Minnesota Experiment. In Simmonds' disease the appearance of edema is frequent though probably less than might be expected in view of the degree of inanition. Edema was found in 18 per cent of the proved cases of Simmonds' disease assembled by Escamilla and Lisser (1942). The fairly frequent (10 to 20 per cent) coincidence of diabetes insipidus with Simmonds' disease may distort the edema tendency. Moss (1942) reported one such patient, a youth of 18, 174 cm. (68.5 in.) tall, whose weight fell to 32.1 kg. (68.8 lbs.) before he died. The plasma protein concentration in this patient was 5.1 gm. per 100 cc. ($A/G = 1.6$); at that protein level edema would be expected to be present. Apparently, however, this patient was not edematous at any time.

Edema has been stated to be unusual in anorexia nervosa except in connection with severe anemia or after successful treatment has started (Berkman *et al.*, 1947). D. J. Stephens (1941) reported a patient with a serum protein concentration of 4.9 gm. per 100 cc. with no edema and said he had been unable to find a single autopsy report in which edema was mentioned. In Gull's (1874) classic paper it was stated that edema may appear in extreme emaciation. Berkman (1930) found 3 cases of pitting edema in a clinical series of 117 patients, but in another series of 25 patients, 12 had edema on admission to the hospital and 4 others developed edema under treatment (Berkman, Weir, and Kepler, 1947). Farquharson (1941) noted that edema appeared very late, if at all, in anorexia nervosa. These differences in comparison with simple semi-starvation seem to be real but are puzzling. Two possible explanations, both of which assume that anorexia nervosa patients are not really resistant to edema, may be suggested

TABLE 403

EDEMA IN ANOREXIA NERVOSA. Summary of findings on admission of a group of patients at the Mayo Clinic, on whom complete serum protein analyses were made and whose body weights were at least 40 per cent below the standard for their height. Edema is graded 0 to 4 where 1+ means least perceptible pitting of the lower legs and 4+ is general anasarca. Protein values are in gm. per 100 cc. of serum.

(Berkman, Weir, and Kepler, 1947.)

Case	Edema	Serum Protein		
		Total	Albumin	Globulin
2.....	2+	6.20	4.40	1.77
3.....	1+	6.39	4.02	2.37
4.....	1+	6.38	4.18	2.20
5.....	2+	5.65	4.42	1.23
6.....	0	6.67	4.85	1.82
7.....	0	5.58	4.00	1.58
9.....	2+	5.27	3.16	2.11
18.....	0	5.68	3.65	2.03
21.....	0	7.72	4.95	2.77
22.....	0	6.49	4.58	1.91
25.....	0	6.49	4.46	2.03

In the first place, it is not impossible that edema is sometimes not looked for, or at least not specifically noted, in the case reports; this might explain the difference in the reported incidence in the two series at the Mayo Clinic (Berkman, 1930; Berkman *et al.*, 1947). In the second place, anorexia nervosa patients tend to have a far smaller water intake than hungry people; they do not "soup" their food with salt and water and sometimes avoid swallowing anything, including water, as much as possible. In any case there seems to be no very close correlation between the level of the plasma proteins and the presence of edema (see Table 403).

Growth and Development

IN TIMES of food shortage there is a strong tendency to sacrifice the adults in favor of the children. Similar behavior is exhibited by many animals. The basic instinct to preserve the race or species seems to be involved. In human society also the pregnant and lactating women generally receive special considerations.

The processes of growth and development require energy and building materials over and above the demands of ordinary maintenance and the cost of physical work. In the adult a severe reduction of food intake results in a diminution of voluntary activity, which decreases the caloric expenditure, and an actual destruction of some tissues already formed, to furnish energy. Both of these adjustments tend to occur also in children and in pregnant and lactating women, but the effects on the formation of newly differentiating tissue requires special examination. To what extent, if any, does the growth of new tissue take precedence over the maintenance of old tissue when the food intake is insufficient for both? Which growth processes are most sensitive to undernutrition? When good feeding is resumed after a period of growth arrest by undernutrition, what is the course and character of the resumption of growth? What, if any, are the long-time residues from undernutrition in the growing child? At what age is sensitivity to undernutrition greatest? These are general problems of both theoretical and practical importance.

Until now systematic, long-range scientific observation and measurement in relation to human nutrition – as in other areas of human biology – has been so deficient that many of the most important and intriguing questions remain unanswered. The faults and weaknesses of the available evidence may be judged from the following review; the obvious omissions represent blank spots in a large but fragmentary body of data. One of the largest problems of medical science has to do with the “degenerative” diseases – arteriosclerosis, the neoplasms, diabetes, and so on. Presumably the bases for these developments are laid down in the earlier life, and quite possibly in the growing period, of the individual. What is the effect of a period of undernutrition in early life on the incidence and time course of these later developments? This is a question on which no real evidence for man can be offered at present.

In default of material obtained during starvation, it is necessary to refer frequently to studies from non-starvation periods and areas in which growth and development were studied in various groups of people subsisting at different levels of nutritional excellence as judged by conventional means. The resulting comparisons, of course, cannot properly differentiate between the effects of bad diet

and those of bad housing, sanitation, care, and heredity. Generally speaking, there is a correlation between all these items and economic circumstance, and many dubious conclusions may be drawn by failure to recognize these complications. Moreover, it should be noted that most of the evidence now available refers to quality rather than quantity of the diet and so is limited in its application to famine conditions.

Length of Gestation and Duration of Labor

There is very little quantitative evidence in the literature pertaining to the effects, if any, that maternal diet may have on the length of the gestation period. Logical explanations for the lack of data are apparent. The recognized difficulty of obtaining accurate and reliable information on the time of conception is an important limitation. The usual obstetrical practice of placing conception at two weeks prior to the date of the first missed menses is, of course, only approximate and may frequently lead to rather gross errors in estimating the duration of pregnancy. In times of famine, when menstruation is commonly irregular, data on the length of gestation are generally unreliable and in most cases worthless. Another difficulty is the large normal interindividual variation in the length of gestation; differences would indeed have to be large before they could be considered of any practical significance.

Balard and Chastrusse (1942) were unable to find any change in the length of gestation in France during the early part of the German occupation in World War II. The estimated mean duration of pregnancy during the first three months of 1938, 1941, and 1942 was 277, 278, and 278 days, respectively.

A positive influence of adequate diet on the course of pregnancy was observed by Ebbs, Tisdall, and Scott (1941) among the 380 pregnancies they carefully followed in Toronto, Canada. The mothers were divided into 3 groups according to the adequacy of their diets. The food intake of the patients in the "poor" diet group was inadequate in both calories and proteins; the "supplemented-to-good" diet was approximately the same as the "poor" diet, plus extra food furnished by the survey group to make the diet adequate; and the "good" diet group had a normal adequate diet. An obstetrician at the prenatal clinic and in the hospital rated the condition and the progress of the patients during the last half of pregnancy.

The average duration of labor was 5 hours shorter in the supplemented-to-good diet group than in the poor diet group. The statistical significance of the 5 hours' difference was not given, nor were sufficient data included to allow calculations of the significance. It was further reported by the obstetrician in charge that the course of labor was good to fair in 76 per cent and poor to bad in 24 per cent of the women in the poor diet group, while 97 per cent of the patients in the supplemented-to-good diet group had labors that were considered good to fair.

In the Harvard School of Public Health survey "no statistically significant relationship was found between the prenatal diet and the duration of labor" (Burke, 1945). The average duration of labor (12 hours) was the same for all primiparae whether in the good or the poor diet group. For the multiparae, la-

bor occupied an average of 8 hours for the good diet group and 12 hours for the poor diet group. There were 4 Caesarean operations in the poor diet group and none in the good diet group. About 50 per cent of the poor diet group and 58 per cent of the good diet group had normal deliveries. The similarity in the percentage of complications of labor in the diet groups is misleading, however, because "it is evident that while the incidence of minor complications was approximately the same in both diet groups, 18 and 14 per cent, respectively, 36 per cent of the poor to very poor groups had major complications, while in the good or excellent diet group only 24 per cent had major complications" (Burke *et al.*, 1943).

An investigation designed to show whether supplementing the maternal diet with additional vitamins and minerals would have any beneficial influence on the course of pregnancy and labor and on the newborn child was conducted in London by the People's League of Health (1946) from March 1938 to the end of 1939. A total of 5022 pregnancies were followed for from 16 or more weeks before delivery to term; of the group, 2510 received a daily vitamin and mineral supplement of iron carbonate, calcium lactate, iodine, manganese, copper, B complex vitamins, vitamin C, and halibut liver oil.

The investigators found "no significant difference between the treated and the controls" for the average duration of labor in all age groups. "A difference of 3.3 hours in favor of the treated which almost approaches significance" was reported for the primiparae 30 years of age or over. The data cited in their Table X, however, did not support that conclusion. For the treated group of 30 years and over the mean duration of labor was 27.3 ± 1.38 hours, and for the control group it was 24.0 ± 1.35 hours. The data presented from the total survey show for the primiparae a progressive increase in the length of labor with increasing age; no correlation between age and duration of labor was found for the multiparae.

Although the evidence is far from clear-cut, it nevertheless points strongly toward the conclusion that moderate variations in the quality of the maternal diet have little if any influence on the duration of labor or the length of the gestation period. There is no useful information on the effect of caloric intake. It must be recognized that the available data are from surveys in which the maternal diet may have been poor but was far from being as restricted as that prevailing under famine conditions. Whether or not extreme dietary restrictions would have any influence is not indicated by any of the data obtained from man.

Toxemias of Pregnancy

In view of the general clinical interest in the influence of maternal nutrition on the prevalence and severity of toxemias of pregnancy (Williams, 1945; Land, 1945), it is surprising that few data or observations on the problem are presented in any of the recent reports from the famine areas of Europe. The lack of data could mean that (1) no observations were made or (2) the incidence of the toxemias during the famine periods was not strikingly different from that occurring during normal times and, consequently, warranted no special mention in the publications. It is hardly conceivable that practically all the careful and

thorough observers making surveys would completely overlook such an important question. It becomes necessary to depend mainly upon the non-starvation, peacetime surveys for information on the role that adequacy of the maternal diet might play in the prevention or alleviation of the toxemias of pregnancy.

Strauss (1935) named hypoproteinemia with a resulting water retention as the primary factor in the etiology of the toxemias of pregnancy. He found the colloid osmotic pressure of the blood to be reduced by about 33 per cent in cases of eclampsia. When fifteen patients with toxemia of pregnancy were given diets containing 260 gm. of protein per day, the symptoms and signs of toxemia gradually disappeared. Five similar pregnant women suffering from toxemia, who were fed diets containing only 20 gm. of protein per day, became decidedly worse during a 2-week period on the experimental diet. Strauss believed that the beneficial results observed in the first group might well have been due to the large protein intake.

In the Toronto, Canada, survey, Ebbs, Tisdall, and Scott (1941) reported no difference in the incidence of pre-eclampsia between the group on the poor diet and that on the good diet. Toxemia was, however, about twice as prevalent in the poor diet group as in either the good or the supplemented-to-good group; the protein and caloric content of the diet for the latter two groups was considerably higher than for the poor group. The small number of cases of toxemia observed in each group does, however, detract from the conclusiveness of the data. Burke *et al.* (1943) and Stuart (1945a), reporting the Harvard School of Public Health survey, stated that the "greater incidence of complications during pregnancy among the women with a poor to very poor diet is due largely to a high incidence of pre-eclampsia in this group." No cases of pre-eclampsia were seen among the "excellent" and "good" diet groups, while 44 per cent of the women whose diet during pregnancy was considered to be "poor to very poor" had pre-eclampsia. There was an 8 per cent incidence of pre-eclampsia in the intermediate diet group. The authors (Burke *et al.*, 1943) concluded that "a significant relationship exists between prenatal nutrition and the incidence of pre-eclampsia during pregnancy." The observed relationship may well have been due to the protein differential of nearly 50 per cent between the excellent and the very poor diet groups.

Williams and Fralin (1942), in a study of 514 pregnancies in Philadelphia, found no relationship between the occurrence of toxemia and the protein intake of the diet. In the group there were 46 cases of mild pre-eclampsia, 6 cases of severe pre-eclampsia, and 8 cases of hypertensive cardiovascular disease. The mean protein intake was 67 gm. per day for the toxemia group and 66 gm. for the group of 392 who had no complications of pregnancy of any kind. A comparison of the normal and toxemia groups on the basis of grams of protein per kilogram of desired body weight, vitamins A and B complex intake, and "other food factors" resulted in no positive influence of the diet on the course of the pregnancies. The British experience with supplementation of the maternal diet with minerals and vitamins indicated a slightly lower, but statistically probably not significant, number of toxemias in the supplemented groups (People's League of Health, 1946; Balfour, 1944).

Luikart (1946) reported the results of using prophylactic methods for the prevention of toxemias in a large number of pregnancies. No cases of toxemia, pre-eclampsia, or eclampsia occurred in 1000 women in whom the body weight increase during pregnancy was managed correctly by the use of a high protein, low calorie diet. Luikart infers that the high protein intake was an important part of the procedure.

It has been reported that there was a decrease in the toxemias of pregnancy in Germany during the "starvation" years of World War I (Editorial, 1917). The more recent emphasis on the role of protein nutrition in the toxemias is scarcely in agreement with reports that a starvation diet — which is by necessity also a low protein diet — may be a factor in *decreasing* the incidence of the toxemias. Smith (1947b) found it "distinctly surprising to learn that all Dutch obstetricians had seen less than the expected amount of toxemia during the hunger period. Review of records from the Midwifery school substantiated the impression." During the hunger months of 1944–45 the percentage of complications diagnosed as toxemia was 1.9 as compared with 3.8 per cent for the first few months after the hunger period and 3.2 and 3.4 per cent, respectively, for 1938–39 and early 1944. Smith pointed out further that "a factor difficult to evaluate was the definite scarcity in table salt available during the hunger period. The concurrent reduction of toxemia suggests that future studies of nutrition and toxemia must be carefully evaluated with regard to salt intake as well as to specific type and duration of maternal food habits. To say that a good diet will reduce the incidence of toxemia and a poor one increase it is futile until we can specify the exact elements constituting goodness and poorness in this regard." A diet which may be rated good for growth or general maintenance purposes may not be the best type of diet during times of special stress such as pregnancy. In any event the relationship of maternal diet to toxemias of pregnancy does not yet appear to be settled.

Congenital Malformations

It has been repeatedly demonstrated in many species of laboratory and domestic animals that certain types of congenital malformations frequently appear in the offspring as a result of maternal nutritional deficiencies (for literature review, see Warkany and Nelson, 1942; Warkany, 1944, 1945). Whether human congenital malformations can be induced by, or are related to, poor diet of the mothers during the course of the pregnancy can never be tested experimentally; the solution to the problem must be sought in careful analysis of records from areas where food intakes have been seriously curtailed. It might be hoped that the experiences in Europe during World Wars I and II would yield some valuable information.

Ivanovsky (1923), in describing the experiences during the Russian famine of 1921–22, remarked that the number of "monsters and children [born] with different anomalies [was] considerably increased." No data were furnished on the number of normal children and children with malformations that were born in the years before the famine and during the famine; the total number of births was, of course, greatly reduced in the famine years. An increase in the incidence of congenital malformation during famine periods does not prove a direct rela-

tionship between the maternal diet and the malformation. During times of famine the entire structure of life is drastically altered, creating factors which in themselves may be responsible for abnormalities in fetal growth.

Burke *et al.* (1943) observed a correlation between the adequacy of the maternal diet and the pediatric rating of the offspring. Almost all the cases of congenital defects were found among the infants born to mothers whose diet during pregnancy had been severely inadequate. Of the 36 mothers in the poor to very poor diet group, 6, or 18 per cent, of the offspring were listed in the class "congenital malformed infants"; there were no congenitally malformed infants from the 31 mothers in the good or excellent diet group (Stuart, 1945a). Two congenital defectives were born to mothers in the fair diet group. It is particularly interesting that 4 of the 8 infants with congenital defects were born to mothers who had pre-eclampsia. Stuart (1945a) pointed out that "pre-eclampsia doesn't occur early in pregnancy, and congenital defects obviously are initiated early in pregnancy. So we can't say that pre-eclampsia was a cause of the congenital defects but it might be that the faulty diet before and during early pregnancy led to the congenital defects as well as to the pre-eclampsia."

The data from animal experiments reported in the literature suggest that "the period during which maternal malnutrition might result in fetal maldevelopment is probably over by the first 6 or 8 weeks after conception in the human. Moreover, it appears likely by the same inference that improvement in human nutrition within 4 weeks after conception would probably allow the fetal organogenesis to proceed normally" (Smith, 1947a). Smith (1947a) found in Rotterdam that there was no significant difference in the incidence of malformation for the prewar period 1938–39, the pre-hunger period 1943–44, the hunger period 1944–45, and the post-hunger period of late 1945; an incidence of about 1.5 per cent was present for all periods. When, however, the data were limited to the infants conceived after hunger had become severe but not later than 4 to 6 weeks before liberation—including just the period when, from animal results, the diet effects might be expected to be greatest—the incidence of malformed was 2.42 per cent and 4.44 per cent in Rotterdam and The Hague, respectively, as contrasted with an average incidence of about 1.5 per cent.

Smith (1947b) pointed out that even though the percentage incidence appeared to be strikingly in favor of a relationship between maternal malnutrition and congenital defects, "the small number of properly timed pregnancies renders the difference of no statistical significance. Frank malformations were encountered in only seven infants, or 3.1 per cent but these seven were not concentrated among the offspring of those women whose diets had been lowest in vitamin A and riboflavin, the two elements of critical importance to proper fetal development in animals." The experimental evidence for a relationship between the vitamin content of the animal diet and the incidence of malformations among the offspring was discussed in detail by Warkany (1944, 1945).

In spite of the lack of statistically convincing evidence for humans, the results from the Harvard School of Public Health survey (Burke *et al.*, 1943; Stuart, 1945a) and from the Holland survey (Smith, 1947a, 1947b) are highly suggestive that normal fetal development can be influenced by the maternal

diet. What the important factors in the diet are cannot be answered from the data available at present. The water soluble vitamins are probably not important factors in European famine because famine diets in that part of the world are generally not low in those vitamins, and Smith's evidence does not incriminate vitamin A. A low calorie famine diet, or for that matter a poor diet in general, is also a low protein diet. The possible effects of the two factors cannot be separated at present.

Miscarriages, Stillbirths, and Premature Births

A question of considerable practical interest is whether the adequacy of the maternal diet has any influence on the course and outcome of pregnancy. Ebbs, Tisdall, and Scott (1941) reported that in the obstetrical history of some 200 multiparous patients, 24 to 39 per cent had had one or more miscarriages, 5 to 13 per cent abortions, 11 to 20 per cent premature births, and 2 to 10 per cent stillbirths. The fact that probably 50 per cent of multiparous patients have experienced one or more major complications in the course of their pregnancies emphasizes the seriousness of the problem and the necessity for a careful investigation of factors that might be important in influencing the frequency of these complications. Several recent reports have attempted to clarify the role that maternal nutrition may have in the normalcy of the course of pregnancy.

TABLE 404
COMPLICATIONS DURING PREGNANCY, as percentage of pregnancies, occurring among women in various diet classes (condensed from Ebbs, Tisdall, and Scott, 1941).

	Poor Diet	Supplemented-to-Good Diet	Good Diet
Threatened miscarriage	8.4	1.1	2.4
Miscarriage	6.0	0.0	1.2
Premature birth	8.0	2.2	3.0
Stillbirth	3.4	0.0	0.6

The incidence of complications, as percentages of the pregnancies, reported by Ebbs, Tisdall, and Scott (1941) for different diet groups, is presented in Table 404. The data indicate that the percentages of miscarriages, threatened miscarriages, premature births, and stillbirths were higher in the poor diet group than in either of the other two groups. In view of the small number of complications of pregnancy in each diet group and the lack of statistical analysis of the data, there is some doubt of the justification for the authors' positive statement that "the incidence of miscarriages, stillbirths and premature births in the women on poor diets was much increased."

In the Harvard School of Public Health series "all stillborn infants, all infants who died within a few days of birth except one . . . all prematures and all functionally immature infants were born to mothers whose diets during pregnancy were very inadequate" (Burke *et al.*, 1943). Of the 216 pregnancies studied, one or more of the quoted complications was present in 15 per cent

By grouping the various types of complications together, a statistically significant relationship was observed between the adequacy of the maternal diet and the course of the pregnancy.

In the People's League of Health survey (1946) the incidence of stillbirths was 2.4 per cent in the treated and 3.1 per cent in the untreated primiparae and 2.0 and 2.2 per cent in the treated and untreated multiparae, respectively; the opposite relationship held for neonatal mortality (death before 8 days), the early death rate being lower in untreated groups. (Treatment consisted of daily supplementation with vitamins and minerals.) On the basis of the 57 stillbirths in the 2510 treated mothers and the 69 stillbirths in the 2512 untreated women, the committee concluded that the treated mothers had a more favorable experience in regard to prematurity and stillbirths than did the untreated group; this conclusion is certainly questionable on the basis of the data presented.

The famine conditions in parts of Europe during the years of World War II furnished "natural" experiments from which to study the influence of maternal diet on complications in pregnancy. Baron and Audry (1942) reported that in the Maternité de Dijon, France, the proportion of stillbirths and neonatal deaths was substantially the same in 1941 as it was before the war, even though the mothers had experienced dietary restriction in both calories and proteins. The restriction, however, was in general not serious enough to be a real threat to the physical condition of the pregnant women.

TABLE 405

INCIDENCE OF UNFAVORABLE TERMINATIONS OF PREGNANCY in Rotterdam, as percentage of conceptions (Smith, 1947).

	Prewar Period 1938-39 (674 births)	Pre-Hunger Period 1943-44 (659 births)	Hunger Period 1944-45 (412 births)	Post-Hunger Period Late 1945 (464 births)
Abortion and miscarriage	1.67	5.6	2.2	3.7
Premature birth	5.27	4.98	6.3	5.15
Stillbirth	3.5	3.2	1.8	2.5
Neonatal death (10 days)	1.55	3.0	2.36	3.0

The outcome of pregnancy was analyzed by Smith (1947a, 1947b) for Rotterdam before, during, and following the severe famine months of late 1944 and early 1945. The data are presented in Table 405. Smith warned that even in normal times statistics on miscarriages and abortions are notoriously untrustworthy and that the identification of prematurity is a problem, particularly when amenorrhea and menstrual irregularities are prevalent. Assessing the data in the light of their limitations, Smith could find no evidence that there was any increase in the incidence of abortion, miscarriages, premature births, stillbirths, or neonatal deaths during the months of the famine. The small reduction in stillbirths observed in Rotterdam during the hunger period was substantiated by mortality statistics from other municipalities furnished by the National Bureau at The Hague. If maternal nutrition is of prime importance in regulating the

outcome of pregnancy, an increased incidence of complications certainly would have been expected in Rotterdam. The total period of serious food shortage was of sufficient duration to cover most of the prenatal life of some of the infants and half or more of the prenatal life of many. The experiences reported by Smith raise serious doubt as to the influence of the quantity of the maternal diet on the course of pregnancy.

The data for the starvation period of the siege of Leningrad (first half of 1942) show a positive relationship between the premature births and stillbirths and the restriction in food intake (Antonov, 1947). The percentage of stillbirths for 1940–42 is given in Table 406. In the first half of 1942 premature births reached 41.2 per cent of all births, while in the second half of 1942, when food was more available to at least some, the proportion was near the normal rate of 6.5 per cent. The proportion of stillbirths was 5.6 per cent in the January–June period of 1942. It decreased to a normal value of 2.5 per cent in the July–December period of 1942. Antonov concluded from his data: “It seems obvious that the cause of the unusually high proportions of premature births and of stillbirths in the first half of 1942 was hunger during pregnancy, that is, the insufficient quantity and the unsatisfactory quality [lack of vitamins] of the women’s food.” The high neonatal mortality of 9 per cent of those born at term and 30.8 per cent of those born prematurely was no doubt largely due to the lack of heat, water, and other facilities during the winter of 1941–42, even though death was “attributed to a certain extent to the low vitality of many of the children.”

TABLE 406
PERCENTAGE OF STILLBIRTHS FOR 6-MONTH PERIODS OF 1940–42; material from the Leningrad State Pediatric Institute (Antonov, 1947).

Year	Number of Live Births		Number of Stillbirths		Percentage of Stillbirths	
	Jan.–June	July–Dec.	Jan.–June	July–Dec.	Jan.–June	July–Dec.
1940.....	2685	1639	72	40	2.73	2.44
1941.....	2007	1049	49	34	2.44	3.24
1942.....	391	77	23	2	5.55	2.53

According to the Famine Inquiry Commission’s report on Bengal (1945), “the falling off in the number of live births during famine is presumably due largely to an increase in the incidence of abortion, miscarriages and stillbirths resulting from malnutrition and disease. It is well known that a woman’s capacity to bear living children is impaired by malnutrition.” Careful consideration of facts would hardly justify such a positive statement except in severe famine conditions where starvation is rapidly progressing toward death. Under the more usual circumstances of semi-starvation where food intakes are restricted (as in occupied Europe during World War II), there is little acceptable evidence that the lesser degree of maternal undernutrition in any way influences the incidence of abortions or miscarriages. The observations in Holland lead Smith (1947b) to remark that “figures for abortions and miscarriages are included to

satisfy the reader's curiosity, but there is no reason to assume that they are accurate or that conclusions can be drawn from them."

Size at Birth

From analyses of birth weights for the years of World Wars I and II, a rather clear concept of the role of maternal nutrition on fetal growth has evolved. There is a general concensus of opinion that the extent of undernutrition experienced in Central and Western Europe and in England from 1914 through 1918 had little or no influence on the size of the newborn infant (Müller, 1918; Lanstein and Rott, 1928; Huggett, 1941; Needham, 1942; Wolman, 1943; Antonov, 1947). Although some slight decrease in birth weights may have occurred in the later part of World War I, the average birth weights were seldom found to be decreased by more than 50 to 100 gm. (Lanstein and Rott, 1928; Antonov, 1947). Sontag, Pyle, and Cape (1935) were unable to establish any correlation between the caloric intakes of the mothers and the weights at birth of the infants for the charity patients at the Miami Valley Hospital in Dayton, Ohio. The average weight and height of the infants were normal (length 50 cm.; weight 7.34 lbs.).

The reports from Russia for World War I did, on the other hand, indicate a suppression of prenatal growth during periods of caloric restrictions; but the point was emphasized that the caloric restrictions must be very severe before any substantial decrease in prenatal growth occurs. Decreases in birth weights of from 160 to 200 gm. (about 5 per cent) were reported by several Russian authors for 1919 as compared with 1911 or 1913 (Antonov, 1947). In Odessa during the famine years of 1921-22 the weights of newborn infants were 113

TABLE 407
BIRTH WEIGHTS OF INFANTS BORN IN ODESSA DURING THE FAMINE YEARS OF 1921-22 AND DURING THE YEARS 1924-26 when food supplies were ample (Gerschenson, 1931).

Birth Weight (kg.)	1921-22		1924-26	
	Number of Infants	% of Total	Number of Infants	% of Total
2.50-3.00	317	35.0	2,020	20.0
3.00-3.50	384	42.5	3,968	39.0
3.50-4.00	165	18.2	2,886	28.3
Over 4.00	39	4.3	1,296	12.7

TABLE 408
BIRTH WEIGHTS AND LENGTHS OF INFANTS GROUPED ACCORDING TO THE PRENATAL DIETARY RATING OF THE MOTHERS (Stuart, 1945a).

Prenatal Dietary Rating of Mother	Birth Weight (kg.)		Birth Length (cm.)	
	Average	Range	Average	Range
Excellent or good	3.86	3.07-5.20	51.8	46.9-54.6
Fair	3.38	1.55-4.18	50.0	45.0-54.4
Poor to very poor	2.64	1.48-4.06	47.2	40.6-52.7

gm. for primiparae and 210 to 258 gm. for multiparae below the weights observed during the prosperous years of 1924-26 (Gerschenson, 1931). The percentage distribution of the infants in the various weight classes for Gerschenson's data is presented in Table 407. In the famine years the incidence of infants weighing 3 kg. or less was nearly twice as high as in the prosperous years, while the incidence of large babies (weights over 4 kg.) was about one third of normal.

The results from the Harvard School of Public Health survey appear to show quite conclusively that the prenatal dietary status of the mother does influence the size of the newborn infant even in peacetime (Burke *et al.*, 1943; Stuart, 1945a). The significant data from the 216 cases are presented in Table 408. The average birth weight was decreased about 12 per cent when the mother's diet dropped from the excellent-to-good class to the fair class. The decrease in weight was even greater (22 per cent) when the fair and the poor-to-very-poor diet classes were compared. The infants from mothers in the lowest dietary classes were on the average only 68 per cent as heavy as those whose mothers had good diets. The same general relationship, except less marked, was present for the body length of the newborn. Of the 216 cases followed, the prenatal diet was considered good or excellent in 31 cases, fair in 149 cases, and poor to very poor in 36 cases. It is apparent from the data in Table 408 that the diet of the mother was not the only factor in determining the size of the infant, since there was considerable overlapping in both weight and length measurements between dietary classes.

Ebbs, Tisdall, and Scott (1941) were unable to find any correlation between average birth weights and the adequacy of the prenatal maternal diets.

Burke *et al.* (1943) also observed a significant relationship between the average daily protein intake of the mothers during pregnancy and the weight and length of the infants at birth. Their essential data are condensed in Table 409. There was an average difference of about 1.2 kg. in the birth weights of the infants of mothers whose diet contained less than 45 gm. of protein per day and those whose diet contained 85 or more gm. The infants whose mothers had less than 45 gm. of protein per day during pregnancy were, for both boys and girls, only about 70 per cent as heavy and 89 per cent as long as the infants whose mothers had 85 or more gm. of protein per day. The relationship be-

TABLE 409
RELATIONSHIP BETWEEN THE AVERAGE BIRTH WEIGHT AND HEIGHT OF
INFANTS AND THE AVERAGE DAILY PROTEIN INTAKE OF THE MOTHERS
during the fourth through the ninth months of pregnancy
(Burke, Harding, and Stuart, 1943).

Protein Intake (gm.)	Birth Weight (kg.)		Birth Length (cm.)	
	Boys	Girls	Boys	Girls
Under 45	2.95	2.67	47.6	46.8
45-54	3.18	3.13	49.3	48.7
55-64	3.38	3.41	50.2	49.9
65-74	3.64	3.52	51.4	50.3
75-84	3.74	3.66	52.0	51.4
85 and over	4.15	3.86	53.3	52.4

tween protein intake and fetal weight would be expected from the data presented in Table 409 because the protein content of the diet was one of the important items used in the classification of the adequacy of the diets.

Data illustrating the influence of food shortages in World War II on the birth weights of children are available for several of the European countries. In France, Baron and Audry (1942) compared birth weights in 1941 with those in 1935-40. In 1941, 32.3 per cent of the newborn weighed less than 3 kg. and 67.7 per cent weighed more as compared with percentages of 26.4 and 73.6, respectively, in 1935-40. Of the children born during the autumn of 1941, Vignes (1942) found that 55 per cent weighed less than 3.3 kg.; this percentage was 46 in 1934-38 and 47 in 1940. In Bordeaux the average weight at birth was 3.306 kg. in 1938, 3.244 kg. in 1941, and 3.149 kg. in the first 3 months of 1942 (Balard and Chastrusse, 1942). The same general relationships held for boys and girls and for the primiparae and the multiparae. The slow decline in the average weight of the newborn in France during 1941 and 1942 appeared to continue into 1945. Trémolières (1947) reported birth weights for 1945 that were below the 1942 values by 60 gm. for boys and 36 gm. for girls. These small differences were considered to be statistically significant. It must be admitted that the changes in birth weights observed in France during World War II were not very striking; in general, food intakes were not severely restricted over long periods of time in most of these areas.

A comparison of the birth weights and lengths in Greece for 1927-28 and 1942 indicates that there was a slight decline during the early part of the occupation (Valaoras, 1946). Food intakes were often severely restricted from early in the occupation (June 1941) until the middle of 1942, when some relief was furnished by the International Red Cross packages. The birth weights for boys and girls, respectively, were 3.5 kg. and 3.2 kg. in 1927-28 and 3.26 kg. and 3.09 kg. in 1942; the average decrease was 240 gm. for the boys and 110 gm. for the girls. Both boys and girls showed only about 1 cm. decrease in average length at birth. A decrease in birth weight of 600 gm. was reported for Vienna between August 1944 and August 1945 (Husslein, 1947).

TABLE 410

APPROXIMATE AVERAGE WEIGHT OF INFANTS BORN IN 8 SUCCESSIVE 6-WEEK PERIODS from October 1, 1944, to October 1, 1945, in Rotterdam and The Hague (Smith, 1947).

Period of Birth	Average Birth Weight (kg.) for Percentile:				
	90	75	50	25	10
1944					
10/1-11/15	3.975	3.725	3.400	3.175	2.910
11/16-12/31	3.910	3.625	3.350	3.075	2.875
1945					
1/1-2/14	3.825	3.500	3.200	3.010	2.810
2/15-3/31	3.725	3.500	3.215	2.975	2.800
4/1-5/15	3.650	3.450	3.210	2.975	2.700
5/16-6/30	3.750	3.550	3.180	2.910	2.650
7/1-8/15	4.000	3.680	3.350	3.100	2.915
8/16-9/30	4.950	3.875	3.480	3.200	2.975

Severe caloric undernutrition was present in Holland during the fall of 1944 and the first 5 months of 1945. The effects of severe maternal caloric restrictions on the size of the infants born during the famine period and for a few months following it have been presented by Smith (1947a, 1947b) and Boerema (1947). Boerema found that the infants born to mothers who had succeeded in obtaining an adequate food intake were on the average 51 cm. in length and weighed 3.800 kg., while the infants born to mothers who had lived on 1500–1800 Cal. per day were only 46.5 cm. in length and weighed 2.650 kg., a difference of 4.5 cm. and 1150 gm. Smith (1947a, 1947b) found a considerably smaller decrease in average birth weight as a result of the widespread starvation. Caloric intakes reached their lowest point (average 1144 Cal. per day) in January 1945; the estimated intake in April 1945 was 1427 Cal. per day. The weight of the newborn decreased as the starvation period continued, but the decline in weight lagged behind the fall in caloric intake. The approximate average birth weights, condensed from Smith's data, for the children born in 8 successive 6-week periods from the fall of 1944 to the fall of 1945 are given in Table 410. Liberation, with an increase in food, occurred in May 1945, but the lightest children were born in June and July of 1945. The birth weights did not return to normal until August and September, at which time the food intake had been restored nearly to normal. Conversely, birth weights did not decrease until after two or three months of the restricted food intake had passed. Smith believed the data warranted the conclusion that severe caloric restriction for the mother decreases the birth weight of the infant but that the effects are most pronounced during the last half or last trimester of pregnancy.

True famine conditions existed in some of the besieged cities of Russia in World War II. Conditions in Leningrad were especially serious from the time of the city's encirclement in September 1941 until the pressure began to ease somewhat in February 1942. The effect of the food restrictions on the size of the newborn have been reported by Antonov (1947) for the Department of Newborn of the Leningrad State Pediatric Institute. Some of the essential data are given in Table 411. The decreased maternal food intake in the last few months of 1941 had very little effect on the birth weight of the infants. However, the average birth weight of infants carried to term in the first half of 1942 was 579 gm. below the 1941 average for boys and 502 gm. for girls. The effects of maternal undernutrition on infant birth weight were also apparent in the second half of 1942, even though the food restrictions were much less severe than during the first part of 1942.

TABLE 411
AVERAGE WEIGHT OF INFANTS BORN AT TERM IN LENINGRAD
(Antonov, 1947).

	Average Weight at Birth (kg.)	
	Boys	Girls
Jan.–June, 1941	3.444	3.302
July–Dec., 1941	3.344	3.222
Jan.–June, 1942	2.815	2.760
July–Dec., 1942	3.199	2.890

In the Snegirev clinic in Leningrad the average decrease in the weight of the newborn in 1942 was 500 gm., and in the obstetric-gynecologic clinic of the Second Leningrad Medical Institute the average birth weights in 1942 were 410 gm. less than in 1940 (cited by Antonov, 1947). Poremsky (cited by Husslein, 1947) reported that birth weights in Leningrad during the siege fell an average of 410 gm.; birth height was affected only when maternal under-nutrition was serious.

The decrease in the average size of the newborn as reported from Leningrad was nearly twice as great as that reported from Holland. In both places true famine conditions were present for probably not more than about 4 months, and serious food restrictions lasted about the same length of time. Although the weight decreases in Leningrad and Holland were not the same, the two reports are in agreement that (1) infant size can be influenced by maternal diet, and (2) the effects of maternal undernutrition on the prenatal growth of the fetus are most pronounced in the last three or four months of pregnancy. Analysis of 3550 births in Chingtu (West China) for the years 1938-45 showed no differences in the average birth weights even though the nutritional status of the mothers was changed markedly by the blockade (Lee, 1948).

Seasonal Growth

Periodicity, the cyclic character of nature, is so consistently encountered that it can be considered one of the laws of nature, and "since man, as an inhabitant of this earthly planet, is a part of the planet and must set himself in equipoise with the world around him, the idea readily suggests itself that periodicity influences certain phenomena of our organism" (Brugsch, 1920, cited by Nylin, 1929). Many of the cyclic activities of man are so commonplace that they need no discussion (e.g. the rhythmic action of the heart and respiration, the menstrual cycle in women, sleep and wakefulness, work and rest, ingestion of food and digestive activities, etc.). Assuming that both man and his natural environment are subject to periodic variations, the important question arises whether the periodicity phenomena in man are inherent within man himself, whether they are secondary responses to the variation in his external environment, or whether they are both primary and secondary characteristics.

A general discussion of the problems of periodicity in man is beyond the limited scope of this section, but because growth and development are important aspects of the human organism and because the role of nutrition and health in the rate of these processes has been emphasized, it is necessary to consider the seasonal factor carefully. If growth and development progress at a constant rate throughout each year from birth to maturity, no special precautions as to season of the year would be required in making measurements designed to determine whether growth was normal over any time interval in a population group. If, however, growth rate varies to any considerable degree with the season of the year, it would then be mandatory that height, weight, and other anthropometric measurements, to be validly comparable, be made during the corresponding months of each year.

There are enough acceptable data in the literature to permit us to decide whether significant seasonal variations occur in the rate of height and weight

increase. Nylin's (1929) monograph, which includes a review of all the European and most of the American literature that appeared up to 1929, and Marshall's (1937) critical review and synthesis of the studies that had been conducted in the United States prior to 1937 adequately present the important information on seasonal variations in the growth rate of children. Later studies strengthen rather than alter the conclusions that might be drawn from these earlier investigations. The bulk of the evidence indicates that growth both in height and in weight does not proceed at a constant rate throughout the year, but varies with the season, and that the season of maximal rate of weight increase is generally associated with a minimal increase in height.

"In autumnno augetur corporis pondus," wrote Sanctorius in 1614 (Nylin, 1929). Early data on seasonal variations in growth rate were reported by Buffon in his *Histoire Naturelle* in 1777. The observations were those of Montbeillard who made semiannual height measurements on his son from birth (in 1759) through the age of 17. No seasonal variation in the rate of height increase was noted until after the age of 5 years, but from then on the maximal increase in height was achieved in April and May and the minimal increase in September, October, and November.

It was a century later before the next references to seasonal growth appeared. Bowditch (1872) found that about 80 per cent of his yearly increase in height occurred during the summer months. Wretling (1878, cited by Nylin, 1929) observed that the rate of weight increase of the pupils in the girls' schools of Gothenburg was considerably greater during the summer vacation months (June-September) than during the school year. He believed that the summer sun had a stimulating effect and school life had a retarding effect on growth.

From daily height and weight measurements of a group of Danish boys over a 4-year period (1882-86), Malling-Hansen (1884, 1886) concluded that periodicity in growth rate was indeed a fact. The rate of body weight increase passed through a maximum period from August to the middle of December, an intermediate period from the middle of December to the end of April, and a minimum period from May through July. The rate of increase in weight during the maximum period was nearly three times as much as during the intermediate period, while there was often an actual weight loss during the minimum period. The most rapid increase in height occurred from April to mid-August, the minimum increase was from the middle of August to the end of November, and the intermediate rate of increase was from December to March. The rate of increase in height in the maximum period was 2.5 times that of the minimum period. In general, the season of maximal increase in weight corresponded to the period of minimal increase in height. Periodic checks on the food intake of the children showed that their food intakes were greatest in March and that their appetites were not maximal during the fall season of their most rapid increase in weight. Malling-Hansen's observations were, in general, confirmed at about the same time by Key (1885), Camerer (1893), Schmid-Monnard (1895), and others (see Nylin, 1929).

Another series of important reports on seasonal variations in the growth rate of school children appeared between 1920 and 1935. Porter (1920) made weight

measurements at monthly or bimonthly intervals on several thousand Boston children over a period of about eight years. A marked seasonal periodicity in weight gain was observed. The average weight gain per month for the group of boys born in 1905 was 0.21 lb. from January through May and 0.77 lb. from June through December. Hunt, Johnson, and Lincoln (1921) found the period from October to February to be most conducive to weight gain for two groups of New York City boys, and height increase was most rapid in the periods of February to June and June to October.

Bleyer (1917), Holt and Fales (1923), Gebhart (1924), Schiotz (1926, see Nylin, 1929), Veeder and Rohlfing (1927), Nylin (1929), Chaney and Justin (1930), Lange (1930), Orr and Clark (1930), Palmer (1933), and Whitacre (1935) reported data which were in general agreement that weight increase was greatest in the fall months and at a minimum in late winter and early spring. A periodicity in height gain for school children was claimed by Holt (1918) and Hunt, Johnson, and Lincoln (1921) but denied by Porter (1920), Emerson (1927), and Whitacre (1935).

Emerson (1927) concluded that even though growth may not proceed at a uniform rate throughout the year, there was no evidence that the periodicity reported by others was dependent on the seasons. He explained any variation in growth as a reflection of general health status; growth increase would be inhibited during periods of illness. This theory is, however, not consistent with the periodicity of growth observed in Australia (Fitt, 1924), where the period of maximum increase in weight is in the early part of the year and the minimum in the later part of the year, which correspond to fall and spring in the Northern Hemisphere.

Nylin (1929) made weight and height measurements on 809 school children in 1926-27 and on 516 in 1928-29. The measurements were made during the months of September, November, February, April, and May. The age range was from 7 through 15 years. The data used in the final analysis included the measurements on only those children who were present for all weighings. Although there were some sex and age variations, the over-all periodicity in growth was pronounced. The height increase exhibited a marked maximum during March-May and a small maximum during November-January; the two minimums occurred during September-November and January-March. The rate of weight increase varied inversely with the height increase; the greatest rate of weight gain occurred in the fall months when height increase was least, and the least gain in weight occurred during the first 4 months of the year. The increase in height was markedly accelerated in a group of 23 boys who were exposed daily to ultraviolet light lamps during the November-January period when the increase in height was normally slow; the increase in weight during the period of treatment was decreased. Apart from the periodicity which could in part be influenced by exposure to sunlight, there appeared to be secondary periodicities. A large or small increase in weight in one period was followed by a small or large increase in height in the next period; the rate of increase in weight in one period appeared to influence the increase in height in the following period, so that a rapid increase in weight was seldom followed by one in height.

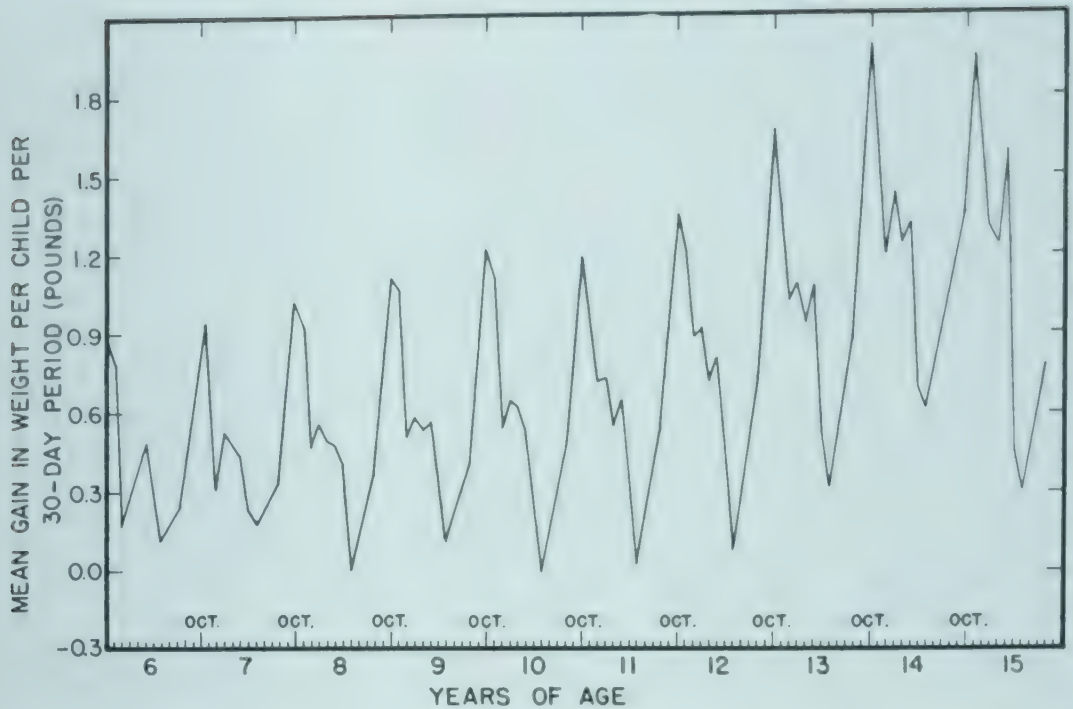


FIGURE 136. SEASONAL VARIATIONS OF AVERAGE MONTHLY GROWTH RATES as observed in yearly age groups of elementary school children in Hagerstown, Maryland (Palmer, 1933).

Lange (1930) observed that preschool children exhibited a periodicity in growth rate. Weight increase was greatest from June through November, intermediate in rate from December to March, and slowest from March through May. Quite similar results were reported by Bleyer (1917) for infants. In the second year of life the infants gained weight at the rate of 86 gm. per day from December through March, 77 gm. per day from April through July, and 113 gm. per day from August through November. Bleyer did not believe that the variations in growth rate could be accounted for on the basis of changes in diet. Orr and Clark (1930) found that the children of Scotland increased their height fastest during the 3-month period from April through June and least during the period from October through December. The increase in weight was fastest from July through September and slowest from April through June, when 25 per cent of the children actually lost weight.

Extensive observations on the seasonal fluctuations of rate of growth were made by Palmer (1933) on a group of 2500 native-born white children, aged 6 to 16, in Hagerstown, Maryland, from 1923 to 1928. The mean gains in weight per month for the various age groups of boys are presented in Figure 136. A distinct seasonal trend is present with the maximum growth rate in October and the minimum in May. The general trend was quite similar for all the age groups studied, the older children showing slightly more marked variations. The essential character of the curve was similar for the boys and girls. Palmer offered no explanation for the seasonal variation in growth rate.

Benjamin (1943) presented later data from England which also showed a seasonal variation in the rate of growth of school children. The maximum in

crease in height occurred in late spring and the minimum in mid-autumn; the maximum weight increase was at the end of autumn and the minimum in mid-spring. In general the seasonal variations were greater in weight than in height. Benjamin pointed out (without giving any evidence) that the true curve of seasonal variation may be affected by changes in a variety of factors such as weather, diet, activity, and sleeping habits.

Allen (1937, 1939) found that in some schools in England there was a variation in growth rate within each school term. The rate of weight and height gain decreased in the final month of the school term. The rates were accelerated during the holidays between terms. The decline in growth rate was greater in the schools where the routine was more rigorous and greater pressure was placed on schoolwork than in the less formal and strict schools.

There is some evidence that the seasons of intrauterine life may also influence the weight at birth and the subsequent development of the child. Needham (1942) cited Toverud (1933) and Abels (1922) as evidence that "infants born during late summer and early autumn [are] significantly heavier than those born during other months." Abels (1922) believed that the greater vitamin intake of the mother in spring and summer had a beneficial effect on the intrauterine growth of the fetus. Hellmuth and Wnorowski (1923) analyzed the birth weights of more than 2000 infants born in the Eppendorfer Klinik between 1919 and 1922 and concluded that the variability in birth weights was so large that small differences in mean weight values would be without meaning, and that, consequently, Abels' conclusion based upon rather limited material was not acceptable. Gerschenson (1931) observed an increase in the percentage of newborn with weights above 3.5 kg. during the months of August, September, and October, with a corresponding decrease in the percentage of infants with birth weights of 2.5 to 3.0 kg. The highest proportion of lighter infants was of those born in February, March, and April. Gerschenson believed there was a relationship between the increased metabolism due to the summer sun and the weight of the newborn. An increased metabolic rate during the summer months and upon exposure to ultraviolet radiation has been demonstrated by others (Nylin, 1929; Lindhard, 1912; Benedict and Finn, 1928). In China, Lee (1948) reported birth weights that were greatest in the winter and spring.

Pintner and Forlano (1939) and Mills (1941) found that the intelligence of children conceived during the winter months was somewhat higher than that of children born during the winter, and Goodenough (1940) explained this difference on the basis that winter conceptions occur more frequently among parents of the higher socioeconomic levels, making the intelligence differences due to hereditary rather than seasonal factors. Held (1940) and Roberts (1944) could find no relationships between the season of conception and the child's intelligence.

Size of Children—Earlier Surveys

The effect of the limitation of food supplies on the size and growth of children during the latter part of World War I and following it received considerable attention from the German investigators of that period. Some of the general aspects of the observations have been reviewed by Wolman (1943), but the data are of sufficient interest and importance to warrant presentation of more of the details.

TABLE 412
COMPARISON WITH PREWAR NORMS OF MEAN WEIGHT AND HEIGHT OF BOYS IN BERLIN
ORPHANAGES IN 1919 (Davidsohn, 1919).

Age (yrs.)	Weight (kg.)			Height (cm.)		
	Prewar	1919	% Diff.	Prewar	1919	% Diff.
2.....	12.7	9.5	25.2	85.0	76.1	10.5
3.....	14.7	11.3	23.1	93.0	82.8	11.0
4.....	16.5	13.5	18.2	99.0	90.0	9.1
5.....	18.0	14.9	17.2	104.0	96.7	7.0
6.....	20.5	16.1	21.5	109.0	98.8	9.4
7.....	23.0	19.9	13.5	115.0	106.8	7.1
8.....	25.0	20.4	18.4	120.0	114.2	4.8
9.....	27.5	21.5	21.8	125.0	117.5	5.0
10.....	30.0	24.6	18.0	130.0	123.8	4.8
11.....	32.5	27.2	16.3	135.0	127.9	5.3
12.....	35.0	29.2	16.6	140.0	132.7	5.2
13.....	37.5	31.5	16.0	145.0	135.2	6.8
14.....	41.0	33.2	19.0	151.0	140.4	7.0

TABLE 413
COMPARISON WITH PREWAR NORMS OF MEAN WEIGHT AND HEIGHT OF GIRLS IN BERLIN
ORPHANAGES IN 1919 (Davidsohn, 1919).

Age (yrs.)	Weight (kg.)			Height (cm.)		
	Prewar	1919	% Diff.	Prewar	1919	% Diff.
2.....	12.2	8.9	27.0	84.0	75.8	9.8
3.....	14.2	11.1	21.8	92.0	82.4	10.4
4.....	15.7	12.6	19.7	98.0	87.6	10.6
5.....	17.0	14.6	14.1	103.0	97.0	5.8
6.....	19.0	15.9	16.3	109.0	100.6	6.0
7.....	21.0	18.7	11.0	113.0	110.7	2.0
8.....	23.0	19.2	10.5	118.0	113.5	3.8
9.....	25.0	21.6	13.6	123.0	118.5	3.7
10.....	27.0	23.2	14.1	128.0	121.8	4.8
11.....	29.0	25.6	11.7	133.0	126.8	4.7
12.....	32.0	27.2	15.0	139.0	130.3	7.6
13.....	37.0	32.0	13.5	146.0	136.8	7.7
14.....	43.0	36.2	15.8	153.0	140.9	6.3

TABLE 414
COMPARISON OF THE WEIGHT AND HEIGHT OF CHILDREN ENTERING SCHOOL IN MUNICH
FOR THE YEARS 1912 AND 1917-18 (Pfaundler, 1919).

		Weight (kg.)				Height (cm.)			
		Prewar	War	Diff.	% Diff.	Prewar	War	Diff.	% Diff.
Age 6									
Girls		18.55	17.49	-1.06	-5.7	109.71	109.94	+0.23	+0.2
Boys		19.21	17.66	-1.55	-8.1	110.90	109.68	-1.22	-1.1
Age 7									
Girls		19.61	19.23	-0.38	-2.0	113.31	112.93	-0.38	-0.3
Boys		20.16	20.19	+0.03	+0.1	114.05	114.59	+0.54	+0.5

Davidsohn (1919) compared the height and weight for sex and age of Berlin orphanage children in 1908–9 with those in 1919. The basic data are given in Tables 412 and 413. The diet of the children during the latter part of World War I was probably less than two thirds optimal in calories, fats, and proteins. In 1919 the average monthly body weight gain was 0.10 kg. for boys and 0.01 kg. for girls, 7 to 14 years of age, as compared to prewar normal gains of 0.20 kg. for boys and 0.30 kg. for girls. The body weights for the various age groups in 1919 were below the 1908–9 norms by 3.0 to 7.8 kg. for the boys and 2.4 to 6.8 kg. for the girls; the percentage weight deficits were 13.5 to 25.2 for the boys and 10.5 to 27.0 for the girls. The decrease of growth rate was less striking in height than in weight. The boys were 5.8 to 10.6 cm. (4.8 to 11.0 per cent) and the girls 2.3 to 11.3 cm. (2.0 to 10.6 per cent) shorter in 1919 than before the war. In 1919 the Pirquet index was low for age and sex groups up to 9 years, after which age the values were comparable to the 1909 values. The deficits represented an average, in terms of normal peacetime growth, of about 1½ years for the boys and 1¼ years for the girls. A comparison of the 1919 measurements with those for 1908–9 underestimated the actual growth deficit because there was a progressive increase in the average body size of German school children between 1908 and the outbreak of war.

In Munich, Pfaundler (1919) compared the height and weight of 2500 school children, 6 and 7 years of age, in 1917–18 with those of 3700 children in 1912 (see Table 414). The average height was only slightly below normal, while the body weight was reduced by 3.9 per cent in 1917–18. Goldstein (1922), on the other hand, reported deficits in body height of children below 6 years of age of from 11 to 17 per cent, with large reductions in body weight. Complete height/weight data for the public school children of Munich for 1921 and before the war have been compared by Martin (1924); the data are condensed in Table 415. In 1921 body weights were from 5.0 to 12.6 per cent below the prewar values for boys and 4.3 to 11.3 per cent below for girls. The heights were reduced by 2.1 to 3.8 per cent and 1.6 to 2.9 per cent for boys and girls, respectively. In spite of the rather severe depression of growth rate in the period through 1921, by 1923, after two years of rather ample supplies, the children were quite comparable in size to the prewar Munich school children. The older

TABLE 415

SIZE OF PUBLIC SCHOOL CHILDREN IN MUNICH BEFORE WORLD WAR I AND IN 1921 (Martin, 1924).

Age (yrs.)	Weight (kg.)				Height (cm.)			
	Boys		Girls		Boys		Girls	
	Prewar	1921	Prewar	1921	Prewar	1921	Prewar	1921
7.....	22.1	21.0	21.7	20.3	120.4	117.0	118.9	116.3
8.....	25.0	23.6	24.1	22.6	126.5	123.9	124.3	121.6
9.....	27.3	25.4	25.8	24.7	131.1	127.7	128.8	126.7
10.....	29.0	26.9	28.2	26.6	134.8	131.2	134.6	130.9
11.....	30.5	28.9	31.9	28.5	137.8	134.7	139.1	135.0
12.....	34.3	31.1	36.3	32.2	143.0	138.9	144.6	140.4
13.....	38.1	33.3	39.1	35.8	148.2	142.5	149.2	145.1

boys in the apprentice groups in 1920 compared favorably with those of 1913 (Kaup, 1921). It should be noted that the dietary reduction in the Munich area in World War I was less than that for many other parts of Central Europe.

The inhibition of growth in children appeared to be less in Frankfurt than in some of the other larger cities of Germany. Schlesinger (1919a, 1919b) found that the average school child in Frankfurt in 1917 was 2 cm. below the 1913 standards. Body weights were below normal, the older children being more underweight than the younger children; the average weight deficits were 0.5 to 1 kg. for 2 to 3 years of age, 1 to 2 kg. for 8 to 10 years of age, and 2 to 5 kg. for children over 12 years of age. The weight deficit was attributed to a loss of body fat and a slowing of growth. It was also observed that a much higher percentage of the children lost weight during each school term in 1917 than in the preceding years. Later observations by Schlesinger (1922) showed a continued depression of growth in 1918, 1919, and 1920. Body weight for age reached its minimum in 1918-19 and for height in 1919-20. In 1918-19 the school children averaged 2 to 3 kg. below the prewar normal and their heights were 3 to 5 cm. below normal in 1919-20. On the average the children were retarded in growth by about 1 year. Rapid improvement occurred in 1921.

TABLE 416
AVERAGE WEIGHT AND HEIGHT OF VIENNESE CHILDREN AT DIFFERENT
AGES COMPARED WITH BRITISH STANDARDS (Gibbon
and Ferguson, 1921).

Age (yrs.)	Weight		Height	
	Lbs.	% Deficit	In.	% Deficit
1-2.....	19.6	14.8	28.5	6.6
2-3.....	20.9	23.7	28.5	13.6
3-4.....	21.9	29.6	30.7	15.8
4-5.....	29.4	16.0	34.0	11.2
5-6.....	33.7	12.8	36.2	11.5
6-7.....	37.5	10.7	38.8	11.2
7-8.....	39.7	14.6	43.4	4.0
8-9.....	42.5	15.8	47.2	(-0.9)
9-10.....	49.8	9.3	48.7	1.0
10-11.....	50.8	17.4	50.8	1.7
12-13.....	62.2	14.8	53.9	2.6
13-14.....	69.6	14.1	56.1	2.0

Consistently large body weight deficits were reported for the children of Vienna following World War I (Gibbon and Ferguson, 1921). Average body weights for age ranged from 10 to 30 per cent below the British standard (see Table 416). Viennese children between 5 and 11 years of age averaged 3.5 lbs. lighter than British children of the same age. The height deficits were less striking (0 to 16 per cent). A comparison of the Viennese and British children is justifiable because the average weight and height of adults in the two populations are very similar. The deficits were comparable to those reported by Davidsohn (1919) for Berlin orphanages.

Size of Children—Later Surveys

Special emphasis has been placed upon the proper use of anthropometric measurements by pediatricians for the evaluation of the nutritional status of children. It is generally agreed that height, weight, and possibly some other anthropometric data are desirable adjuncts to clinical examinations of children, but because "there has been no standard practice as to the measurements taken, the techniques adopted, the norms used for reference, or the evaluation of the data obtained," the value of such data has been limited (Vickers and Stuart, 1943). Many of the data concerning growth and development of children as related to their nutritional status suffer seriously from the limitations stressed by Vickers and Stuart (1943). That such limitations are inherent in the techniques of measurement and interpretation is not necessarily the case. By means of the "grid" method, described in detail by Wetzel (1941, 1943a, 1943b, 1943c), signs of malnutrition can be recognized long before the more usual clinical signs appear. The whole process of interpretation is placed upon an objective basis. Unfortunately, the Wetzel "grid" technique has not been used in any of the nutritional surveys relevant to the present problem. The more usual subjective methods of interpretation have, however, permitted broad generalizations about the relationship between general nutritional status and normalcy of growth and development in children.

Reports from England indicated that growth was essentially normal during the war period up to 1945. Billington, McCance, and Widdowson (1943) found no significant difference in height and weight for boys in two schools for the years 1939 to 1943. Marrack (1947a) reported evidence that there was a small decrease in both the height and the weight of boys 13 years of age in 1941 as compared with prewar standards; the decrease was rectified by early 1942. There is good evidence, however, that the school term interfered with optimal growth both before and during the war years, even though the yearly gain was normal (Allen, 1937, 1939; Widdowson and McCance, 1944). In most cases the rate of weight increase was about twice as fast during the holiday period as in the school term. It is apparent from these observations that incorrect conclusions might be drawn from weight comparisons over a period of time if the measurements were not made at comparable times in relation to time of year and school term.

There is evidence from nutritional surveys in the United States and Canada that some relationship exists between body size of children and nutritional status. Emerson (1934) found that "30 to 50 per cent of all children are malnourished"; in this case malnourished meant, among other things, that the children were at least 7 per cent underweight. Palmer (1935) observed that children from low income homes were smaller than children from comfortable income homes. During the depression of the 1930s the relative weights for children from comfortable income homes in 1928–30 fell when they became low income homes in 1930–33. The sons of the professional and managerial classes in the United States were found to be 3 per cent taller and 6 per cent heavier than the sons of unskilled workers (Meredith, 1941); it is well recognized that the adequacy of the diet increases in the higher income groups. Armattoe (1943) also reported

underweight and underheight children in the poor districts of Toronto, but the height weight ratio was normal. From an intensive study of a few underdeveloped children, Talbot *et al.* (1947) concluded that "viewed as a whole, [the] clinical, nutritional and metabolic studies coupled with data in the literature suggest that retarded growth in the thin patients . . . was due primarily to caloric malnutrition."

The influence of wartime nutrition on the growth of French children has been discussed in considerable detail. Weight increments for the school year of 1941-42 were reported for 23,000 children in Paris by Cayla *et al.* (1942) and Launay *et al.* (1942). Of this group, 12.8 per cent lost weight (more than 300 gm.) and 21.2 per cent did not gain weight during the year, as compared with a normal incidence of no weight gain in only 0.5 to 2 per cent of school children in 1939. The percentage of children who failed to gain weight was greater in the poorer districts of Paris. The older children (adolescents) also appeared to have suffered relatively greater deprivation than did the younger groups. Gounelle, Vallette, and Moine (1942) measured the height and weight in 1941 of 1075 Parisian boys and girls ranging in age from 3 to 15 years, and compared the findings with 1935 standards. In general, there was in 1941 a deficit in height of 1 to 5.5 cm. for the boys and 1.5 to 2.0 cm. for the girls. The average weight deficit was from 1 to 2.28 kg. for boys and 0 to 1.3 kg. for girls; the smallest deviations from normal were in the 5- to 8-year group.

Comparable data were reported by Aubertin (1942) for other groups of Paris children. Weight increases for 1941-42 and 1938-39 were compared for children living in the better and poorer districts of Paris. In 1938-39 only 0.73 per cent of the school children lost weight during the school year. For the school year 1941-42 3.3 per cent of the children lost weight in the better districts and 10.1 per cent lost weight in the poorer districts. The percentage of those who lost weight increased with age; 8.2 per cent of the 6- to 7-year-old boys and 16.6 per cent of the 14- to 15-year-old boys lost weight. The same trends held for the girls.

The general impression of the doctors of Paris, based on the examination of some 28,000 children, was that the restrictions of food supply in 1941-42 retarded the growth rate of children. Weight was affected more than height and older children more than younger children (Huber *et al.*, 1942). In 1941-42 the school children of Marseilles were slightly shorter and lighter than Boston children, but the height weight ratios were about the same (Stuart and Kuhlmann, 1942). In unoccupied France, however, body weights were about 14 per cent below the standards for Iowa children, while heights were only 7 per cent less. No explanation was offered for the apparent differences in the height weight ratio in Marseilles and in the unoccupied areas of France.

The depression of growth rate in the children of France appeared to be relieved somewhat during the year 1942-43. Hedon (1946) reported quite normal body weights and heights for Paris children up to the age of 12 to 13 years. The children of 14 to 15 years had a weight deficit of about 2 kg. for the girls and 3 kg. for boys. The percentage of boys who lost weight was 12 to 18 in 1942 and 5 to 12 in 1943, and for the girls the percentages were 12 to 20 and 5 to 12 in 1942 and 1943, respectively (Poulain, 1943). The decrease in the incidence of

weight loss in 1943 was associated with a slightly better food intake. Stuart (1945b) estimated that the average caloric intake for children 6 to 13 years of age in Marseilles was 1600 Cal. in 1942 and 1725 Cal. in 1943. The protein, vitamin (except vitamin D), and mineral content of the diet was probably sufficient to meet minimal requirements in 1943. Vaucher and Violle (1943) reported that the children 4 to 9 years of age examined in the Centres Sanitaires de L'Enfance in 1943 were above the Paris norms for before the war, but over 10 years of age they were below prewar Paris norms. In spite of the lack of a weight deficit these children were not as tall as the prewar Paris children. This is one of the few reports indicating that height might have been affected more than weight.

Some recovery from the weight and height deficits occurred in France in 1944. Boulanger-Pilet and Breuille (1946) found that 3.8 per cent of the boys and 2.8 per cent of the girls in Paris schools had lost weight in 1944; these are less than half the percentages that lost weight in 1943 (Poulain, 1943). The average weight deficit in 1944 as compared with 1938 was 2.4 kg. for boys and 1.3 kg. for girls (Boulanger-Pilet, 1946). A general improvement in growth rate was also reported by Trémolières (1946). The most complete comparison of the weight and height of Paris school children in 1938 and 1944 was made by Laporte (1946). Some of the data are presented in Table 417. On the average the

TABLE 417

WEIGHT AND HEIGHT AVERAGES OF PARIS SCHOOL CHILDREN OF DIFFERENT AGES
IN 1938 AND 1944 (Laporte, 1946).

Age (yrs.)	N	Weight (lbs.)			Height (in.)		
		1938	1944	Diff.	1938	1944	Diff.
Boys							
6.....	17	47.9	40.6	-7.3	45.7	43.2	-2.5
7.....	242	48.4	47.8	-0.6	46.2	45.5	-0.7
8.....	37	56.5	52.4	-4.1	49.4	47.6	-1.8
9.....	243	59.3	56.3	-3.0	49.4	49.2	-0.2
10.....	47	66.0	62.7	-3.3	52.5	52.2	-0.3
11.....	262	71.4	68.0	-3.4	56.2	53.1	-3.1
12.....	44	83.2	75.3	-7.9	56.7	55.2	-1.5
13.....	212	85.5	81.4	-4.1	57.6	55.6	-2.0
14.....	26	101.5	90.1	-11.4	61.3	59.7	-1.6
15.....	139	113.0	105.3	-7.7	63.2	62.4	-0.8
16.....	30	135.5	125.9	-9.6	66.1	65.5	-0.6
Girls							
6.....	14	43.6	41.7	-1.9	44.0	43.3	-0.7
7.....	234	46.9	45.5	-1.4	44.8	43.3	-1.5
8.....	35	55.4	52.1	-3.3	48.3	46.9	-1.4
9.....	237	56.1	53.4	-2.7	49.0	47.8	-1.2
10.....	44	69.9	62.8	-7.1	53.3	52.0	-1.3
11.....	260	72.2	69.3	-2.9	54.1	53.5	-0.6
12.....	41	86.7	79.2	-7.5	57.8	56.3	-1.5
13.....	223	90.6	87.6	-3.0	58.3	57.8	-0.5
14.....	39	103.7	101.4	-2.3	60.6	59.5	-1.1
15.....	134	106.5	104.1	-2.4	61.3	60.3	-1.0
16.....	30	120.2	124.5	+4.3	62.2	63.0	+0.8

boys were 2.7 kg. and the girls 1.5 kg. lighter in 1944 than in 1938; heights were also less in 1944 in both boys and girls in all age groups, except in the 16-year-old girls. Weight for equal height remained quite constant. A constant height-weight index is not the usual experience in times of underfeeding; the rate of increase in weight is generally affected more than the increase in height.

TABLE 418

WEIGHT AND HEIGHT AVERAGES FOR BELGIAN CHILDREN FOR THE YEARS 1938-39, 1942, 1943, and 1944 (Ellis, 1945).

	1938-39		1942		1943		1944	
	Wt. (kg.)	Ht. (cm.)	Wt. (kg.)	Ht. (cm.)	Wt. (kg.)	Ht. (cm.)	Wt. (kg.)	Ht. (cm.)
Age 7-8								
Boys	24.2	125.0	21.1	118.9	21.7	119.9	22.2	121.0
Girls	23.8	122.2	20.7	119.1	21.5	119.7	21.7	120.6
Age 9-10								
Boys	27.3	132.1	26.5	130.8	26.1	129.0	26.8	131.0
Girls	26.4	130.0	25.6	129.5	25.9	129.0	25.5	128.4
Age 11-12								
Boys	34.2	142.8	30.1	137.4	30.9	138.8	32.1	139.0
Girls	36.0	144.0	31.9	140.0	30.7	137.9	31.1	138.0
Age 13-14								
Boys	41.0	151.0	37.1	149.1	36.8	147.6	38.8	147.7
Girls	44.0	151.0	38.4	149.5	38.1	146.9	41.7	149.9

A detailed comparison of the height and weight of children in urban Belgium for the years 1938-39, 1942, 1943, and 1944 has been made by Ellis (1945). Table 418 condenses the essential data for all the school children examined in Anderlecht from September 1942 through 1944. Heights and weights for all age and sex groups showed an increase in 1943 over 1942, and there was a further increase in 1944. The 1944 values, on the average, were still about 2 to 4 cm. and 1 to 2 kg. below the 1938-39 averages. Height for equal weight was slightly greater in 1942 than in 1944, indicating that in 1942 the children were underweight even for their height. The general trend since 1942 toward regaining the 1938-39 height and weight norms seems to indicate that no permanent decrease in the stature of the children will result from the early wartime food shortages.

Owing to special efforts and precautions by the authorities in charge, little actual starvation was observed among the children of Holland except in the first few months of 1945. Jonxis (1946) stated that "during the whole starvation period of 1945 the rations for babies were the best obtainable. They got enough protein, and the total caloric value of their food was sufficient. For children older than 1 year circumstances were far more difficult. . . . Nearly all these children lost weight, and although in the beginning their height continued to increase, in the last months before liberation this increase also stopped." It must be remembered that during the starvation months when even height increase ceased, the caloric value of the diet declined to extremely low levels and there was a degree of undernutrition far more severe than that seen in the other

TABLE 419

WEIGHT AND HEIGHT AVERAGES FOR AGE GROUPS FROM SURVEYS IN OFFENBACH, FRANKFURT, AND KASSEL IN OCTOBER 1947 (Personal communication).

Age (yrs.)	Males			Females		
	Wt. (lbs.)	Ht. (in.)	Wt. Deviation from Standard (%)	Wt. (lbs.)	Ht. (in.)	Wt. Deviation from Standard (%)
1.....	24.2	30.8	+7.8	23.1	29.6	+10.5
2.....	29.9	35.2	+2.0	27.5	34.8	-3.8
3.....	33.0	38.2	0.0	33.4	38.5	0.0
4.....	35.9	40.3	+1.4	36.7	40.4	+3.7
5.....	41.4	43.8	+2.7	40.3	42.7	+4.7
6.....	45.8	45.5	+4.6	44.7	45.9	+0.7
7.....	50.2	47.4	+3.7	47.3	47.3	-1.5
8.....	53.2	49.6	-3.6	51.3	49.0	-3.6
9.....	61.2	51.4	-2.7	58.5	51.4	-6.7
10.....	61.4	52.7	-9.7	61.6	52.9	-9.9
11.....	70.0	54.7	-6.9	69.5	54.6	-7.3
12.....	75.5	56.6	-9.5	75.1	56.5	-7.8
13.....	83.4	58.0	-6.0	86.7	59.4	-7.3
14.....	92.8	60.6	-6.3	99.2	61.6	-4.4
15.....	107.6	63.4	-2.4	110.0	63.2	+1.0
16.....	124.5	66.0	+2.0	119.5	63.7	+4.6

occupied countries of Europe. De Haas and Posthuma (1946) found that by the end of 1944 children of school age were 2 years and preschool children 1 year below normal in weight for age, but they were only slightly retarded in growth in height.

Body weight and height measurements were made on all age groups of the population in the American occupied zone of Germany in 1946 and 1947. The condensed data for the children of Offenbach, Frankfurt, and Kassel for October 1947 are presented in Table 419; approximately 50 measurements are included in each age group. The data presented are very similar to those for Württemberg-Baden and Hesse, where about 1.5 million measurements were made in May and June 1947. It was only in the 9- to 15-year-old children that there was any recorded body weight deficit. In these age groups weights of 3 to 10 per cent below what was considered to be "minimal for the maintenance of health" were reported. Actually the weights presented equal the average body weights of Munich school children before World War I and exceed by some 5 to 13 per cent the average body weights for age of the Munich school children in 1921 (see Table 415). There was, of course, a general increase in size for age of children between World Wars I and II, so the data presented in Table 419 do represent true body weight deficits. The implication, however, that these deficits are serious enough to jeopardize the health of the German children of 9 through 14 years of age can hardly be accepted. The weights used as the minimal standard for health more probably represent average normal values.

Depression of growth rate was present among the children of the poor families of Spain in 1941. Robinson, Janney, and Grande (1942a, 1942b) found that 55

to 75 per cent of the boys and 76 to 87 per cent of the girls had body weights at least 7.5 per cent below the standards for Boston school children; normal Spanish standards were not available. Pelvic and chest circumferences for age were about the same in the Madrid and Boston children. It was estimated that 70 per cent of the children under 13 years of age had lost weight, 47 per cent had scant subcutaneous fat, and 19 per cent had poor physical development. Food consumption records indicated average caloric intakes for these families that were "far below maintenance levels."

Metcoff and McQueeny (1946) examined Italian children of refugee, institutionalized, school, and population-at-large groups in 1945. The groups were weighted in favor of those in which malnutrition might be expected. The data showed no marked deviation in height or weight from prewar Italian standards for preschool and school age children; the values were lower than United States norms.

Pyke (1945) was unable to demonstrate any significant changes as a result of the war in the height and weight of 10-year-old children in Vienna. In Greece, however, there were indications of a deteriorating growth rate (and nutritional status) from 1941 through 1944. Valaoras (1946) presented data based on the measurements of more than 100,000 children. According to the Pelidisi index, 15 per cent of 1- to 2-year-old children and 55 per cent of 3- to 8-year-old children were underweight (for height) in 1942 and 1943. In 1944 the percentage of underweight increased to 27 and 64 for the 1- to 2-year and 3- to 8-year groups, respectively.

The severe starvation among the victims in the Warsaw Ghetto apparently had an effect on the growth of the children. Braude-Heller, Rotbalsam, and Elbinger (1946) mentioned in their report that the children failed to gain weight but that the growth in height was affected less. Puberty was also delayed.

Wilson and Mitra (1938) made an interesting report on the relationship between body size and caloric intake for peoples of different regions of India. The caloric value of the diet was about 500 Cal. less in Assam than in Calcutta. The Assam diet also was very low in total and animal protein. The average body size of adults was 10 kg. and 3 to 5 in. less in Assam than in Calcutta. In Barasat, where the caloric intake was intermediate between those of Assam and Calcutta, the body size was also intermediate. The absolute weight increase of the children per year, however, did not differ greatly between regions (2.10 kg., 2.05 kg., and 2.46 kg. for Assam, Barasat, and Calcutta, respectively), nor did the percentage increase in weight per year. The handgrip per unit of body weight was higher among the Assam population than in the other two regions where the diet was better. In spite of the good correlation between caloric intake and body size in the three regions, there is no evidence that the people in Assam are smaller as a result of the lower food intake. The reverse situation may well be the case. The same relationship between food intake and body size has been reported by Tang and Chang (1939) for several regions in China.

There can be no doubt from the evidence in the literature that the growth of children can be and is influenced by a restriction in the food intake. The extent of the growth deficit is related to the severity of the undernutrition. The rate

of body weight increase is much more readily affected than is growth in height, but even height growth can be seriously inhibited when the caloric deficit becomes extreme.

Growth on Refeeding

A problem of even greater importance is whether the growth of a child that has been slowed down by a limited food intake will be accelerated and the deficit corrected once a liberal food intake is provided. There is some evidence, particularly from Germany after World War I, suggesting that the growth depressions produced by food restrictions may not result in a permanent deficit (Wolman, 1943; Keys, 1948).

Martin (1924) concluded that by 1923 the children in Munich were again normal in size and in the course of three years had made up the growth deficit that had occurred during the last years of the war. Abderhalden (1920) described the weight and height changes in a group of underweight 8- to 14-year-old children who were rehabilitated. In a period of 6 to 8 weeks the average weight gain was 3.2 kg. and the height gain 2.8 cm. A few weeks of growth at that rate would more than neutralize the deficit. Block (1920) reported similar experiences in undernourished children sent to Switzerland. In about a month's time the children gained 5.3 kg. of weight, and their total body weights soon reached normal for age and height. Goldstein (1922) recorded weight increases of 8 kg. in 14 weeks when underweight children were refed. In many of the children these increases were greater than the weight deficits before refeeding.

Wolff (1930, 1935) compared the size of German children born before and during the World War I period of food shortages at the time they entered and left school (approximately 6 and 14 years of age). The effects of the postwar food shortages were apparent in the small size of the children entering school in 1924 to 1926 (children who were born in 1917 to 1920). By the time the children finished school (1930–33) the retardation of growth was no longer apparent. "These older children born in the war years have made up their growth deficit. A permanent injury did not result." Actually the children born in 1917 to 1920 were considerably taller and heavier at 14 years of age than children born before the war — an expression of the tendency toward an increase in the size of school children which seems to be characteristic of the present period of history. Wolff found that from 1924 to 1932 body weight had increased 1.0 to 1.5 kg. for children entering school and 4.0 to 5.5 kg. for children leaving school; the height increase was 6 to 6.5 cm. and 6 to 8 cm. for those entering and leaving school, respectively.

The rapid recovery of the body size deficits of 1917–20 led Wolff to take to task those who were bewailing the degeneration and decay of civilization as a result of the depression of the 1930s. The retarded children in Holland made rapid recoveries when liberated and sent to Australia for rehabilitation (De Haas and Posthuma, 1946). From past experience it can be safely assumed that the food crises of the 1940s in many parts of the world, if they are not of too long duration, will probably have no permanent harmful effect on the generation of growing children.

Infectious Diseases and Undernutrition

FROM time immemorial famine and pestilence have been considered an inseparable pair, the twin fruits of war, but inseparable also when famine occurs without man's being the sole creator of his misfortune. Prominent among the diseases thus associated with famine have been scarlet fever, diphtheria, dysentery, typhoid, typhus, cholera, and tuberculosis. Not only have these at times become epidemic in periods of famine, but there is much evidence that their course becomes more severe. It has been rather generally assumed that both the increased incidence and the virulence in such cases are due, at least in part, to the prevailing state of undernutrition. Usually, of course, where famine and severe undernutrition prevail there are present numerous other factors which would tend to spread infectious diseases.

Tuberculosis has received special attention in this connection and is therefore separately discussed in Chapter 47. Here other infectious diseases may be discussed together on the principle that writings about them show in common a superabundance of opinion and a paucity of really pertinent and satisfactory information about their relationship to nutrition. Unnecessary discussion may be avoided at the outset by eliminating all consideration of the supposed effects of specific vitamin deficiencies on susceptibility or response to infection.

The vast majority of statistics on mortality or morbidity from infectious diseases in famine or war conditions are useless in attempting to discover the role of nutrition. As we shall presently see, the most recent evidence of this sort is chiefly of interest in proving that epidemics can be prevented or controlled without any particular attention to diet. But the questions remain. Are undernourished persons peculiarly susceptible or lacking in resistance to infectious diseases? Once the infection is contracted, is the course of the disease much altered in the undernourished person? Are there differences between the various infectious diseases in these respects? What is the mechanism whereby resistance to infection may be altered by a change in the general nutritional state?

Following exposure to or contact with a given infectious organism, the subsequent developments will be determined by the properties of the limiting membranes (skin, mucous membrane), the presence of natural or previously acquired immunity, phagocytosis, antibody production, the circulation, and the physical and chemical characteristics of the host tissues and fluids which must afford the medium for growth of the invading organism. All of these may be altered, directly or indirectly, by undernutrition, but evidence on these several points leaves much to be desired, even in animal experiments.

An outstanding result of various animal experiments is the demonstration that undernutrition has different effects with different infecting agents and different animal species. Decreased resistance or increased susceptibility has often been reported or claimed in undernourished or malnourished animals. But other effects have also been demonstrated. Robinson and Siegel (1944) found that inanition had no effect on the resistance of rats to induced pneumococcal lobar pneumonia. Foster *et al.* (1944) placed mice on a diet reduced to 40 per cent of their normal intake and found increased resistance to the poliomyelitis virus. Low protein diets may delay the onset of symptoms in mice after inoculation with poliomyelitis virus without changing the eventual percentage of mortality (Jones *et al.*, 1946).

The Limiting Membranes

The body temperature of severely undernourished animals, including man, is subnormal. Although the depression of the rectal temperature may be only 1° or 2° F., the abnormality of temperature of the more peripheral tissues may be much greater. The skin temperature certainly tends to be quite low, especially in cold weather. We may safely predict that all the more peripheral tissues will be relatively cold; this follows from a consideration of the heat exchanges as well as the fact that slight peripheral cyanosis is common in severely undernourished persons. The situation at the limiting membranes, then, is that the temperature is less than normal and the local circulation is relatively depressed. The local metabolism of such tissues must be even lower than the general metabolism because of the decreased temperature and circulation. It may be recalled that in the Minnesota Experiment we found the respiratory ventilation to be relatively increased in semi-starvation. This means that the temperature of the nasopharynx must tend to be more than usually colder than the internal tissues and that the number of air-borne organisms brought to the nasopharynx would tend to be relatively large in comparison with the metabolism.

These conditions might well be favorable to prolonged survival of infectious organisms and to their penetration of the limiting membranes to gain entrance to the blood and the tissues generally. The widespread belief that this is the case is perhaps reasonable, though there is little acceptable direct experimental evidence.

There is reason to believe that the epithelial lining of the gastrointestinal tract becomes more permeable to microorganisms in severe undernutrition. The morphological changes in the intestinal tract would strongly suggest this. Ficker (1906) produced evidence to indicate such increased permeability to invasion in fasting rabbits, mice, rats, cats, and dogs. Bacteria, either indigenous to the intestinal tract or experimentally introduced there, appeared in the blood stream and lymph after different lengths of time, depending on the animal species. Three days of fasting sufficed to allow this phenomenon to occur in rabbits, but in dogs several weeks were required. Morgulis (1923, p. 192) pointed out that the failure of Meltzer and Norris (1899) to observe this phenomenon in dogs could be explained by the fact that the animals were fasted only 5 days. According to Mingazzini (1900) the starving animal shows a remarkable accumulation of leucocytes in and near the intestinal wall.

Antibodies — Earlier Studies

Many of the older experimental studies concerning the effect of nutrition on antibodies may be criticized on technical grounds, but some of them are at least suggestive. Konstantsoff (1912) sensitized guinea pigs with different proteins and recorded the anaphylactic response to later injections of the same proteins. Partly starved animals resisted second injections many times larger than those sufficient to produce anaphylactic shock and death in control (well-fed) animals. Konstantsoff also believed that the complement content of the blood decreased during starvation. These straightforward experiments on anaphylaxis seem to leave no doubt that starvation can have a great effect on this phenomenon in guinea pigs. Comparable studies on prolonged undernutrition would be of much interest.

Early studies on agglutinins did not show great changes induced by undernutrition. Ficker (1906) observed no change in the agglutinating power of the serum of rabbits when the animals were deprived of food for a few days. But total fasting and chronic undernutrition are, as we have noted repeatedly, markedly different in many ways. A much more pertinent, as well as technically more satisfactory, study was made by Zilva (1919). He found no disturbance in the production of agglutinins, of complement, and of amboceptor in growing guinea pigs when the diet was restricted for from 10 to 28 weeks. The dietary reduction was such that the restricted animals gained only about 10 per cent in body weight while the controls gained as much as 300 per cent.

Zilva also made similar experiments with diets containing different percentages of proteins. At levels of 8, 12, and 20 per cent of casein in the diet the rate of growth showed the familiar relationship to the protein level, but the ability to produce agglutinins and amboceptor was not affected. In all these experiments Zilva used heat-killed typhoid organisms of the Simpson strain and made considerable efforts to obtain reliable quantitative measures.

Antibody Production — Later Work

The work of Paul Cannon and his associates at Chicago has stimulated much interest in nutritional status and the ability to produce antibodies; their work

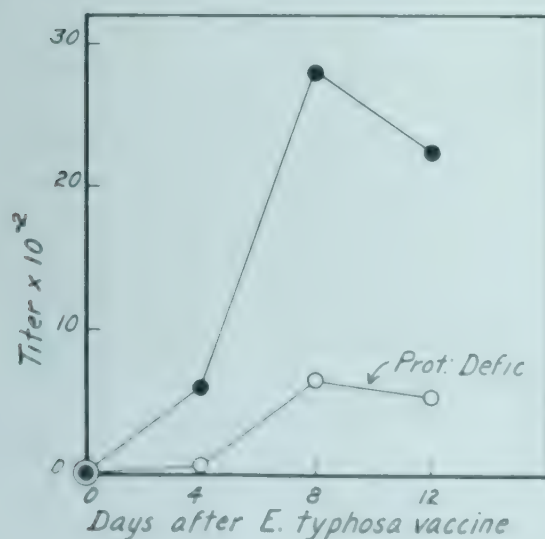


FIGURE 137. AGGLUTININ TITERS OF SERUM FROM YOUNG RABBITS AFTER SUBCUTANEOUS INJECTION OF 0.2 CC. OF A SUSPENSION OF *Eberthella typhosa* VACCINE; means for 8 protein-starved rabbits (average weight 849 gm.) and for 8 well-fed rabbits (average weight 2284 gm.) (data from Cannon, Chase, and Wissler, 1943).

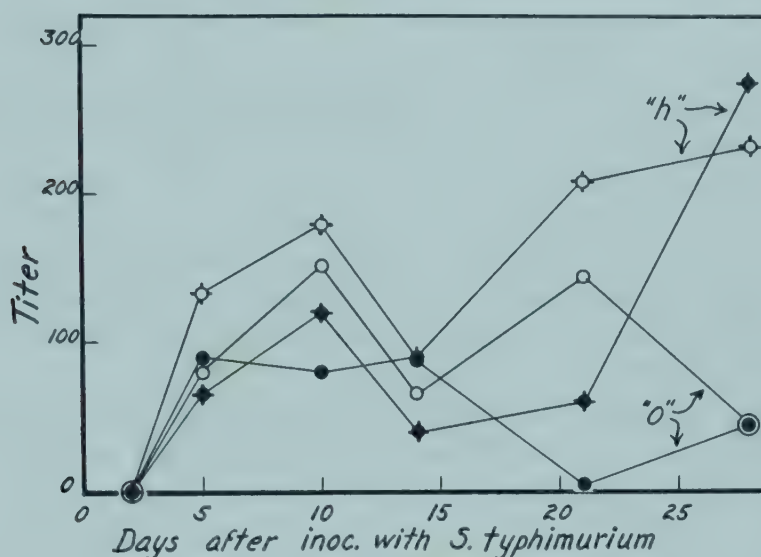


FIGURE 138. MEAN SOMATIC ("O") AND FLAGELLAR ("h") TITERS, AS RECIPROCAL, TO ANTIGENS OF *Salmonella typhimurium* after inoculation of protein-deficient (2 per cent protein in diet) and well-fed (18 per cent protein in diet) young rats. Open circles = means for protein-deficient rats; solid circles = means for well-fed rats. (Data from Metcalf *et al.*, 1948.)

has conclusively demonstrated, in some experimental situations, a relationship between protein nutriture and antibody production. Cannon has repeatedly emphasized the implications of these findings for all undernourished states, particularly for famine (Cannon, 1943, 1944a, 1944b, 1945). The data summarized in Figure 137 are rather typical of the evidence offered by the Chicago group for their contention that protein nutrition is of the highest importance for resistance to infectious disease. The production of agglutinins after immunization with *Eberthella typhosa*, as well as with paratyphoid A and other antigenic agents, was markedly lower in the protein-deficient animals.

The degree of protein deficiency involved in the Chicago studies was extreme, and this aspect of the nutritional state has been the focus of attention. But it is proper to point out that in most cases there was general deficiency as well as protein deficiency and that at the time of testing the disparity between the protein-deficient animals and their controls was even more marked in body weight than in plasma protein concentration. The adult protein-deficient animals suffered body weight losses of more than 30 per cent and the growing animals were almost completely arrested in growth, while their controls were doubling or tripling their initial body weights. The criticism that plasmapheresis was often used to intensify the protein depletion may be leveled at some of these studies, but in later studies this procedure was abandoned with no great effect on the general picture (Cannon *et al.*, 1944; Wissler *et al.*, 1946).

Quite different results have been reported from experiments on rats by Metcalf *et al.* (1948, 1949). The findings in regard to agglutinin titers after inoculation with *Salmonella typhimurium* are summarized in Figure 138. No relationship between protein nutrition and antibody production could be discerned,

though one group of animals subsisted on only 2 per cent protein while the other group had 18 per cent protein in its diet.

The discrepancy between the reports of Cannon's group and those of Metcalf *et al.* and of Zilva (1919) is so considerable that extrapolation to the case of undernourished man cannot be made with any degree of safety. One must agree with Cannon that, ultimately, antibodies are proteins and as such must, also ultimately, be derived from the dietary protein. But there is still uncertainty as to what point of protein deficiency is critical. From the animal experiments so far reported it might appear that only prolonged and extreme deficiency may be consequential.

Some of the later studies on vitamin deficiencies suggest that investigations of immune bodies in malnutrition can lead to erroneous conclusions about resistance and susceptibility. Doan (1946) found that monkeys made leucopenic on diets deficient in the vitamin B complex were very susceptible to infection with influenza virus and hemolytic *Streptococcus* Group C, organisms to which they are ordinarily quite resistant. This was in spite of the fact that these monkeys developed specific precipitins, opsonins, and antistreptolysin titers just as well as did the control animals.

Studies at Wuppertal in 1946

Experimental studies on antibody formation were carried out at Wuppertal in the German Ruhr in 1946 by Gell (1948). In spite of the limited nature of this work, the use of controls and quantitative methods on severely undernourished men gives it unique significance. There were two groups of undernourished subjects: (1) 32 cases of malnutrition from the Barmen Municipal Hospital, and (2) 25 civilian prisoners from the Siegburg jail. Sixteen subjects in a good state of nutrition served as controls. All these persons were given injections of three antigens for which preformed immunity could not be present: (1) tobacco mosaic virus, (2) avian red cells, and (3) a saprophytic vibrio; the last-named failed to produce demonstrable agglutinins. The results for the other two are summarized in Table 420.

The data indicated a statistically significant superiority of the controls over the undernourished persons at all periods and for both antigens. The author's conclusions are based on three major points: "(1) The extremely severe degree

TABLE 420
ANTIBODY FORMATION IN SEMI-STARVED MEN, in response to injections of tobacco mosaic virus and avian red cells. The data presented are percentages of serums giving agglutination at a dilution of 1 to 8 or more, before injection (Before), during 3 weeks after the first injection (3 Weeks), and during 2 weeks after the second injection (Second) (Gell, 1948a).

	Percentages of Serums Giving Agglutination					
	With Avian Cells			With Tobacco Mosaic		
	Before	3 Weeks	Second	Before	3 Weeks	Second
25 prisoners	8.0	60.0	60.0	0.0	52.0	48.0
32 patients	6.3	37.5	65.5	0.0	53.1	31.3
16 controls	0.0	81.3	87.5	0.0	93.7	93.7

of undernutrition from which the test subjects were suffering — severe enough to render active life impossible; and (2) in spite of this, the comparatively small differences between these literally famished subjects and the controls, in first-class condition; (3) the significant fact that there actually has not been any widespread epidemic disease in Germany since the end of the war. These points taken together suggest that undernutrition does not play as large a part in widespread epidemics as is generally supposed" (Gell, 1948a, p. 324).

Gell (1948b) further pointed out that statistical analysis of the Wuppertal data showed a complete failure to correlate antibody production with age, with the serum protein level, or even with the estimated weight loss. These studies at Wuppertal, along with consideration of the complex of factors concerned in the broad question of nutritional state and "resistance" (cf. Schneider, 1946; Parry, 1948), again emphasize the points that generalization is impossible, that practical predictions of the results of famine must be restricted to particular states of nutrition and to particular infective agents, and that the response of one species (man) cannot be gauged safely by the response of other animal species.

Antibodies and the Gamma Globulin

It now seems reasonably certain that the gamma globulin fraction of the blood plasma or serum, as defined by electrophoresis, contains most of the antibody materials so far recognized (cf. Enders, 1944). Chow *et al.* (1945) studied six dogs that were rendered hypoproteinemic by a low protein diet and plasmapheresis; the gamma globulin fraction was reduced in four dogs, unchanged in one, and increased in one. While such evidence suggests that the gamma globulin fraction may be reduced by measures producing general hypoproteinemia, it scarcely proves a direct and perfect relationship between total protein and the gamma globulin fraction under such conditions. It may also be noted that there is no reason to believe that the gamma globulin fraction is made up exclusively of antibodies. In other words, it is not safe to assume that total antibodies may be estimated from the total amount of gamma globulin. At least some antibodies are found in protein fractions other than the gamma globulin (E. L. Smith, 1946).

There is extremely little information about the amount or concentration of gamma globulin in undernourished human beings. Krebs (1946) reported a low level of this protein fraction in the plasma of an undernourished girl; there was no mention of susceptibility to infection. In the Minnesota Experiment there was, on the average, a moderate reduction in the gamma globulin concentration in the blood plasma. We have discussed this in detail in Chapter 20; there was no evidence to associate this change with any alteration in resistance or susceptibility to infection.

Response to Experimental Infection

The first experimental work of consequence on the effect of undernutrition on immunity was reported by Canalis and Morpurgo in 1890. It was known that rats and pigeons have considerable natural immunity against the bacillus of anthrax; Canalis and Morpurgo undertook to see if this would be altered by fasting. The results were negative with rats but a dramatic change was produced in

pigeons. Whereas only 16 per cent of normal pigeons died of anthrax after inoculation, practically all the starved birds died. It was also found that feeding the pigeons during the incubation period would save them unless the starvation prior to inoculation had lasted more than a week. These findings were confirmed by London (1896), who also reported a similar loss of resistance to anthrax in pigeons when the birds were not completely starved but were fed one-fourth rations; very severe food restriction was required, however, and the effect was not seen on one-third rations. London also showed that water deprivation was even more effective than food restriction in producing this loss of resistance.

It is surprising to discover how little systematic experimental work on these questions has been done in the half-century since the work of London. The reports are few and unsatisfactory, and the various workers have reported discordant results with methods that are probably not properly comparable. Robertson and Tisdall (1939) found increased mortality from rat typhoid (*Salmonella mueritidis*) in rats fed several diets, each deficient in various vitamins, minerals, or animal proteins; the absence of all details on food consumption, body weight, and other elementary questions makes it difficult to evaluate this superficial work. In contrast, Edwards (1937) found that the susceptibility of rats, guinea pigs, and hedgehogs to the virus of hoof and mouth disease was not increased when the animals were underfed. Edwards concluded that the experiments "confirm the observations of others that foot and mouth disease infection is most severe in well-grown and well-nourished animals" (p. 1051). Somewhat similarly, Crowe (1937) reported that rabbits which were losing weight on an overcooked cereal diet showed a reduced tendency to develop arthritis when injected with "arthrotropic" streptococci. Crowe suggested that the diet was deficient in vitamins and that the lack of arthritic development reflected a faulty ability of the tissues to react. Sako (1942) reported that the survival time of rats after injections with a virulent pneumococcus was affected by the diet but that the relationships were complicated and no clear undernutrition was involved.

Schneider (1946), after noting the remarkable paucity of reports on the effect of caloric undernutrition on natural resistance to infection, pointed out that most of the recent information comes from experiments on vitamin deficiencies in which caloric undernutrition controls have been run incidentally. In this way Foster *et al.* (1944) found that restricting the intake of the stock diet of mice to 40 per cent of the usual consumption prolonged the incubation time of poliomyelitis infection. This is similar to the delaying effect of a low protein diet on the onset of symptoms, which, however, did not affect the eventual mortality in poliomyelitis (Jones *et al.*, 1946). Seeler and Ott (1944) reduced the food intake of chicks to half their normal level and found that their survival to *Plasmodium lophurae* infection was decreased to 50 per cent. However, studies with rats infected with Type I pneumococcus (Robinson and Siegel, 1944) and with mice infected with *Salmonella enteritidis* (Schneider, 1946) showed no influence of differences in the caloric nutritional state on survival.

In some instances the resistance of animals to experimental infection is definitely worsened by attempts to improve the nutritional status. In mice inoculated with the virus of swine influenza by Sprunt (1948), the lowest mortality

was in the animals on a protein-deficient diet, additions of methionine to the diet resulted in a higher mortality, and the highest death rate was in the animals on the "best" diet with abundant proteins.

Guggenheim and Buechler (1946, 1947) demonstrated the interrelationships between calories and proteins in the resistance of rats and mice to *Salmonella* infection produced by experimental inoculation. In the first place, they found little difference between protein-deficient rats and well-nourished controls in the response to the infection. But rats starved for calories showed some degree of lessened resistance; in these animals the protein intake had little importance. In mice they found that resistance to *Salmonella* was decreased on a low protein, low calorie diet, but this could be at least partly offset by increasing the dietary intake of either protein or calories.

These few examples illustrate the complexity of these questions as well as the relative lack of serious investigation of them. Besides the question of the response to infection in a state of undernutrition, there is the problem of the situation during recovery from undernutrition. Roger and Josué (1900) reported that when coli bacilli were injected into rabbits from 3 to 11 days following a preliminary fast of 5 to 7 days, the animals exhibited markedly greater resistance to the infection. We are not aware of any more recent investigations of this nature.

Natural Infection in the Presence of Undernutrition

The evidence on the effect of undernutrition per se on infections under natural conditions is likewise unsatisfactory, though there is a voluminous literature of opinion and inference. The frequent outbreaks of epidemics in times of famine may conceivably be explained as due to other factors besides undernutrition. It has been noted frequently that the mortality rate of the infectious diseases tends to be greater than normal under such circumstances, but this is not necessarily an indication of lessened resistance in the host organism. There is the possibility that unusually virulent strains of the infecting organisms are involved, the probability that the size or frequency of the inoculum is greater than in ordinary times, and the certainty that medical care is below standard.

Observations on animals have produced a variety of results. In one of the more recent reports, McCay (1942) found that rats on low caloric intakes were unusually free from the ordinary infectious diseases that occur in laboratories. This is reminiscent of Edwards' (1937) opinion about the natural incidence of blackleg and anthrax in cattle and braxy in sheep. In opposition, Allison *et al.* (1946) stated that their hypoproteinemic dogs "become very susceptible to disease" but provided no data for their very small group of animals. Observations such as these have their interest, but from them to application in man is a large and dubious step.

Increased susceptibility to infections does not seem to be a problem in anorexia nervosa or in Simmonds' disease in spite of the fact that these patients may proceed to the most extreme limits of cachexia and may actually die of starvation.

Comparison of mortality from infectious diseases in Europe in World Wars

I and II provides abundant evidence that undernutrition, though perhaps important by itself, is often of far less consequence than other factors. Cowell (1937) remarked that "The great influenza pandemic of 1918-19 appeared to ravage the comparatively well-fed troops as severely as the less well-fed civilian population" (p. 1040). Indeed, many physicians in Central Europe were of the opinion that the mortality among the well-nourished patients was even higher than among those who had been less well fed.

From a practical viewpoint the concentration of attention in recent years on sanitation, disinfection, lousicides, and so on has been eminently justified. It is significant that there were no real epidemics in western Holland or in Greece at the times (1945 and 1942, respectively) when there was mass starvation in those countries. It is also significant that the diphtheria epidemic in Northern Europe in 1946 did not spare, in either incidence or mortality, the American occupation troops, though the contrast between their nutritional state and that of the German inhabitants was very marked.

There are even suggestions that undernutrition provides protection against some infectious diseases. Markowski (1945) observed that in the German prisoner-of-war camp at Hammarstein typhus was attended by a 30 per cent mortality among the Russian prisoners, but that among the German guards the mortality was close to 100 per cent. The Russians were existing under the worst imaginable conditions of nutrition, housing, and sanitation. It was facetiously suggested that the infective lice found the Russians too thin to support an overwhelming infection.

Minor skin infections, especially furunculosis, are generally very common in famine areas and were noted in the undernourished prisoners of the Japanese in World War II (Gottlieb, 1946). Lack of soap and bad sanitation would seem adequate to account for this phenomenon. In the Shinagawa prisoner-of-war hospital in Tokyo the patients were almost uniformly very badly nourished and suffered from vitamin deficiencies as well as caloric starvation. Gottlieb commented on the results in surgery: "Excellent results were obtained in abdominal surgery, even though there was very little asepsis, for our home-made sterilizer was inadequate and our gloves were merely dipped in creosote solution plus the fact that both the air and the operating room were constantly being contaminated by dust and dirt. . . . There was no postoperative peritonitis, very little abdominal distention, and no stitch abscesses appeared."

Uehlinger (1948) observed that in the concentration camp victims seen by the Swiss doctors there was increased morbidity from all enteral infections of the dysentery type, from pulmonary tuberculosis, and from pyodermias, but there was no increase in endocarditis, rheumatic infections, glomerular nephritis and other kidney infections, or in hematogenous tuberculosis. One can suggest that, in general, those infections that primarily involve the mucocutaneous barriers were increased, and that good reasons for this can be advanced without suggesting that "resistance" of the body is involved. On the other hand, infections which require blood transport and internal lodgment in the body would seem not to be increased either in incidence or in severity by a general state of severe undernutrition.

Infectious Disease in Children—World War II

The reports from all the "stress" areas of Europe in World War II are in agreement that children suffered from the pyodermias, furunculosis, and diarrheal diseases which afflicted the starved adult populations in those same areas. The diarrheal diseases were particularly fateful for the infants. In Budapest in 1945 the University Hospital "lost roughly 10 times as many infants with diarrhea" as in the prewar period (Kerpel-Fronius, 1947).

For the skin infections it is not possible to rule out nutritional factors, but the majority of authorities were inclined to blame the bad general sanitation and personal hygiene; lack of soap and hot water alone was a big factor. For the diarrheal conditions the apportionment of blame is difficult because of the fact that in Budapest, in Warsaw (Braude-Heller *et al.*, 1946), and elsewhere there was no proof that infective agents were involved, and it is conceivable that the condition was a primary result of starvation and coarse and strange food. But if infective agents were responsible, there is no need to suggest that the children were unusually susceptible or had a lowered resistance to the hypothetical organisms. As Kerpel-Fronius says (1947, p. 248), the great increase in diarrheal disease in infants seems to be explained by such circumstances as were present in Budapest: "The disintegration of our milk collecting system, the breaking down of the communication system, the lack of ice, and the invasion of flies breeding in billions in the mountains of garbage and wreckage blocking the streets." It was necessary "to disregard a good many hygienic considerations because of the overcrowding of the partially destroyed hospital" (*ibid.*, p. 244). In 1942 there were 527 children admitted; in 1945 the number rose to 1053, and these infants averaged 26 per cent below normal weight in spite of the great prevalence of edema among them. Of those admitted to the hospital 46 per cent died. But there were no epidemics of the usual contagious diseases, and the death rates from pneumonia, empyema, and otitis were even lower than in 1942.

The situation in Budapest had many counterparts elsewhere. That in the Warsaw Ghetto was far more tragic and prolonged. From late 1939 until the final extermination in mid-1942 there was a large population in all degrees of starvation, and new lots of victims, ranging from well-fed to extremely cachectic, were almost daily thrust within the barricades. Almost 300,000 persons died there or were taken out to be murdered by the Germans. Surprisingly good studies were kept in progress on the state of health, and a children's hospital was in full operation. Among both children and adults epidemics were very rare and, when they occurred, ran a benign course, but exanthematous typhus was endemic throughout 2 years. It is interesting to note that typhus deaths diminished in the period when starvation was at its worst (January to June 1942) (Apfelbaum *et al.*, 1946).

Braude-Heller, Rotbalsam, and Elbinger (1946) devoted their attention to the ghetto's children. Skin infections were very common but were attributed to faults of hygiene and did not seem difficult to manage. The physicians were extremely surprised to have very little trouble with contagious diseases (other than typhus) or with infection in general, in spite of low standards of hospital hygiene and large numbers of "imported" infectious cases. In observing groups

of children in rather different states of nutrition, they repeatedly noted that infectious diseases were more frequent and severe in the better nourished children; this was especially marked in the case of measles. Severe epidemics of measles attacked well-nourished evacuees from Germany, but the children in very bad nutritional states rarely contracted the disease. The experience with meningitis was similar. Other points observed were the remarkable absence of allergic reactions, with almost no cases of serum sickness, and the practical disappearance of rheumatic fever. Before the war rheumatic fever had always been rather prominently represented in the children's hospital; in 30 months of semi-starvation there were only 5 cases, 2 of which were recurrences of earlier attacks. This seems almost incredible but it is emphatically stated to have been the case.

The Clinical Picture in Infectious Disease

Caloric undernutrition undoubtedly modifies the clinical picture in infectious diseases in many ways, but the subject has not been studied critically. It must be remembered that the starved person without infection exhibits bradycardia, hypothermia, and leucopenia. The clinical interpretation of heart rates, body temperatures, and leucocyte counts is complicated, therefore, when infection occurs. In 36 famine victims with tuberculosis the average leucocyte count was 11,000, and in 30 of these men the average sedimentation rate was high—52 mm. in 60 minutes (Larson, 1946). In acute pulmonary infections, as well as in tuberculosis in famine victims, Chortis (1946) found tachycardia and fever. This is contrary to the older report of Gerhartz (1917), who stated that starving men with pneumonia may have bradycardia and hypothermia. In India, famine victims in the most extreme degrees of emaciation exhibit fever (102° to 104° F.) when they have malaria or amebic dysentery (Charlton, 1946).

The temperature question is further complicated by the fact that there are indications of relative poikilothermia in starving men; the body temperature is unusually low in cold weather and may possibly tend to be unusually high in very hot weather. This could explain the difference in the reports from India as contrasted with those from Central Europe. However, among the semi-starved Dutch both in Europe and in Java there was a marked lack of febrile response to infection (Netherlands Red Cross Feeding Team, 1948). In Java it was observed that there were many cases of "feverless malaria," in which great quantities of parasites were readily seen in the blood but no fever resulted.

There seems to be rather general agreement that the course of infectious disease, once established, is apt to be unfavorable in the severely undernourished person. Objective data, however, are few, and one must suspect that the conclusion is often drawn simply because it seems a reasonable idea that the general disability of undernutrition would add to the ill effects of infection. Kundratitz (1947) believed that measles was unusually virulent, with an enhanced tendency to relapse, in Vienna during the worst period of food shortage (1945). But we have noted that at Warsaw measles had an unusually mild course in semi-starved children (Braude-Heller *et al.*, 1946). The data on diphtheria in Vienna are more impressive. In 1944 there were 2741 cases with a 5 per cent mortality, in 1945 there were 2880 cases with a 10 per cent mortality; in 1946 the incidence

was higher (3736 cases), but the mortality dropped to 4.6 per cent (Kundratitz, 1947).

Respiratory Infections in the Minnesota Experiment

In the Minnesota Experiment the subjects were housed together with an equal number of young men, also volunteers from Civilian Public Service, who were assigned to the Laboratory in other capacities. With the exception of food, the living conditions of the two groups were identical. Neither group was cut off from outside contacts, which in general they shared. Besides the Laboratory Staff and numerous visitors, both groups of men went to university classes, lectures, concerts, religious services, and movies and had numerous parties at the Laboratory to which girls were invited. There were, then, suitable bases for a comparison of the incidence and severity of upper respiratory infections in the two groups. Records were kept on this score. For the 6 months of semi-starvation there was a total average of 1.1 "colds" per man in the experimental group. Eight colds started at the very beginning of semi-starvation, so that the true incidence of upper respiratory infections during the period of semi-starvation was approximately one cold per man, and, with one exception, these all occurred in the period from February through May (i.e., in the first 4 months of semi-starvation). For comparison, there was an average of 1.2 colds per person in the control group during the same period. Clearly the incidence of colds was not elevated in the undernutrition period.

The severity of the upper respiratory infections was not perceptibly different in the starved men as compared with the control group. Except during February, the first month of semi-starvation, there were no colds graded as severe in either group. There was one difference, however. In 7 of the experimental subjects there developed during the second half of semi-starvation a mild, nonproductive bronchial cough which persisted for a week or more after all other symptoms of the cold had disappeared. This was not disabling or particularly distressing to the men but was apprehensively watched by the Staff physicians. In each case the condition gradually cleared spontaneously without leaving any discernible residue. No such cough appeared in any of the control men.

It may be noted that a mild bronchitis was observed in many cases of war edema in Germany and elsewhere during World War I. Schittenhelm and Schlecht (1919) observed that of several hundred civilians in Germany between the ages of 19 and 33 about half had a persistent cough but no other signs of respiratory infection. Similar observations were made in Europe in World War II. Among the men released from the Mauthausen concentration camp, every second one was coughing, but the coughs were not productive, even in those who had tuberculosis (Hottinger, 1948). At least part of the difficulty may be ascribed to a relative cessation of mucous secretion in the semi-starved man.

The reported situation in Japan is of some interest, though internal evidence in the reports casts grave doubt on the "facts" adduced for the actual nutritional state (U.S. Strategic Bombing Survey, 1947). We are told that World War II produced a progressive and important reduction in caloric intake, and specific figures are given for factory workers indicating average declines in caloric con-

sumption of from 700 to 1000 Cal. daily (*ibid.*, p. 71). But records of the body weights of these workers indicate little or no change over the same years. In any case, the data on infectious disease morbidity and mortality in Japan show no adverse change except in tuberculosis. This would seem to be the more notable in view of the fact that the war produced extremely adverse effects on sanitation and public health control in Japan (*ibid.*).

Diet Therapy in Infectious Disease

We cannot here discuss the question of diet therapy in the presence of infection. There has been much progress since the days of the dictum "Starve a fever and stuff a cold," but it would be less than honest to suggest that practice rests on solid scientific facts in all cases. As a matter of fact, in the British Army undernourished patients were often "therapeutically" starved further if they happened to be febrile; Manifold (1946) stated: "I fear that the old teaching of light diets for fever cases dies hard." There is no dissent from the approval of a good diet, though there can be controversy about just what is a good diet; caloric adequacy or more is always recommended. The virtues of high caloric diets in typhoid fever and infectious hepatitis are unquestioned. We are unaware of any infection for which a calorically deficient diet is generally recommended.

Tuberculosis

TUBERCULOSIS and malnutrition have been associated in the minds of most investigators ever since tuberculosis was recognized as a clinical entity. It is common knowledge that tuberculosis increases in frequency in times of famine, that it tends to progress more rapidly in malnourished patients, and that the disease is often arrested by the simple measures of rest and good food. However, until recently it has been extraordinarily hard to establish exact relationships or even to prove the correctness of the common belief that undernutrition directly favors the development of tuberculosis. There are three principal reasons for this: first, research on tuberculosis has been hampered by the lack of an experimental animal that responds like man to the tuberculous infection; second, tuberculosis tends to heal spontaneously, and there is great individual variation in this respect; finally, wherever there are famine and undernutrition there are also, in the great majority of cases, other important factors which operate to increase the morbidity and mortality from tuberculosis.

Evidence concerning the relationship of tuberculosis to undernutrition has been obtained from four major sources: (1) studies on the incidence and development of tuberculosis among populations existing under famine conditions; (2) clinical observations on the frequency, virulence, and response to diet of tuberculosis in poor and undernourished patients; (3) studies on the metabolism and peculiarities of the tuberculous patient; and (4) experimental attempts to study these questions in animals.

Perhaps the most significant evidence is derived from the study of populations in famine. As far back as accurate records go, tuberculosis has increased in most periods of food restriction. Before discussing this evidence, however, it is necessary to note the complication produced by general trends in tuberculosis morbidity and mortality. Since about 1880 there has been a gradual and progressive decline of tuberculosis mortality in many countries, particularly in Europe. There has been much speculation about the cause of this improvement, and a review will not be attempted here. Many factors are probably intimately concerned, including improved personal and public sanitation, better housing and nutrition, isolation of infectious cases, earlier diagnosis, and an increase in resistance to, or a decrease in the virulence of, the organism. Except in a few specific areas, it is impossible to emphasize any single one of these factors. But, in any case, it is essential to allow for this long-time trend in evaluating the data for any particular period.

World War I—The World Picture

During and immediately after World War I different areas of the world showed quite different changes in tuberculosis mortality, and in general these seem to have been reasonably closely correlated with the nutritional situations in those areas. It is illuminating to compare some of the largest cities, as in Table 421. Cities where there was no real food shortage—New York, Rio de Janeiro, Sydney, Tokyo, Bombay—continued to show a decline in tuberculosis mortality from 1913 to 1920. Cities where there was only a slight to moderate food shortage in 1917—London, Paris, Amsterdam, Rome—showed a moderate rise in tuberculosis mortality in 1917 which tended to return to normal by 1920. Cities where the food shortage was serious—Berlin, Vienna, Budapest, Warsaw—suffered marked increases in tuberculosis mortality. In Leningrad the worst food shortage continued for several years after the war, and tuberculosis mortality was much greater there in 1920 than in 1917.

For some of the worst famine areas of World War I the chaotic conditions allow only incomplete computations of tuberculosis mortality. In 1920 the death rate from tuberculosis, per 100,000, was 504 in Leningrad (Wedenskaya, 1926) and 688 in Kiev (Drolet, 1945). Extreme conditions prevailed in Poland in 1917 with tuberculosis mortalities of 974 per 100,000 in Warsaw and 908 in Krakow.

The data given in Table 421 suggest that the war had no effect on tuberculosis in regions removed from the actual zone of conflict and definite blockade. This is not completely accurate. The interdependence of nations in food, fer-

TABLE 421
TUBERCULOSIS MORTALITY PER 100,000, BEFORE (1913), DURING (1917), AND AFTER (1920) WORLD WAR I. Cities in Group A had no real shortages of food due to the war; cities in Group B suffered minor shortages; cities in Group C had widespread under-nutrition in 1917. Budapest, Leningrad, and Warsaw were still seriously underfed in 1920. All forms of tuberculosis were represented in all the cities except Leningrad, for which only the pulmonary type was listed. (Drolet, 1945.)

	1913	1917		1920	
	Rate	Rate	% 1913	Rate	% 1913
Group A					
New York	199	188	94	126	63
Rio de Janeiro	417	392	94	394	94
Sydney	82	55	67	71	87
Tokyo	427	381	89	352	82
Bombay	219	216	99	187	85
Group B					
London	165	211	128	129	78
Paris	385	358	93	272	71
Amsterdam	154	203	132	157	102
Rome	224	281	125	238	106
Group C					
Berlin	185	323	175	176	95
Vienna	323	479	148	405	125
Budapest	361	639	177	448	124
Leningrad	286	331	116	504	176
Warsaw	306	974	318	337	110

tilizer, and agricultural implements is complex, and the food supplies actually consumed are related to other economic circumstances. It is significant that by the end of the war there were increases in tuberculosis in Scandinavia, Spain, and even Japan (Drolet, 1945).

Germany and Austria—World War I

Kirchner (1921) summarized the data for Prussia from 1876 to 1919; they show a progressive, almost linear decline from a mortality rate, per 100,000 population, of 310 in 1886 to 136 in 1913. It is not clear to what extent this change may be credited to the provision of sanatoriums and other public health measures; there were simultaneous major improvements in housing, nutrition, and working conditions, and a general gain in health (Figures 139 and 140).

There was a small increase in tuberculosis mortality in Prussia in 1914, including 5 months of World War I, but this upswing was no greater than had occurred to interrupt the general decline in 1903, 1899–1900, and 1893. But in 1915 the rise was unmistakable, and for 1916, 1917, and 1918 the mortality rate from tuberculosis rapidly rose to a peak of 230 per 100,000, receding in 1919 to 215. It is interesting to note that another but much smaller rise in tuberculosis mortality occurred in Germany as a whole in 1923 and 1924; this was coincident with the economic chaos and widespread distress associated with the final collapse of the German currency.

A part of the wartime increase in tuberculosis mortality in Prussia as well as in other parts of Germany might well be attributed to the early death of patients with advanced tuberculosis, hastened by the shortage of nurses and doctors, qualitative changes in the diet, and other factors apart from general undernutrition. But all observers agreed that there was a marked increase in new cases and that the course of the disease quickly became fulminating, with rapid dissemination of the infection, tubercular pneumonia, miliary spread, and death within a few months (Anon., 1924). There was also a great increase in morbidity. Bruns (1920) quoted the records from Breslau, which showed 4277 new cases in the year 1917–18 as compared with 2906 in 1913–14.

The greater part of the increased tuberculosis mortality in Germany occurred in the cities (Selter and Nehring, 1921; Kirchner, 1921). In Germany, as elsewhere, the urban tuberculosis death rate has always exceeded that of the rural areas. Before the war the urban rate had been 35.8 per cent greater than the rural rate, but at the peak in 1918 this difference had risen to about 56 per cent. Kirchner's (1921) calculations for the rates of change in successive years are marred by arithmetical errors, but recalculations from his raw data show the progressive development of the urban disadvantage, as well as the fact that the maximal rate of increase occurred in 1917 in both urban and rural districts (see Table 422).

A comparison of conditions in urban and rural districts in Germany is significant. The German rural population is peculiar in that the people do not live on their farms but rather in crowded villages, where housing, sanitation, and public health facilities are inferior to those in the cities. Food, however, was always relatively plentiful in the rural areas and remained so during the war years

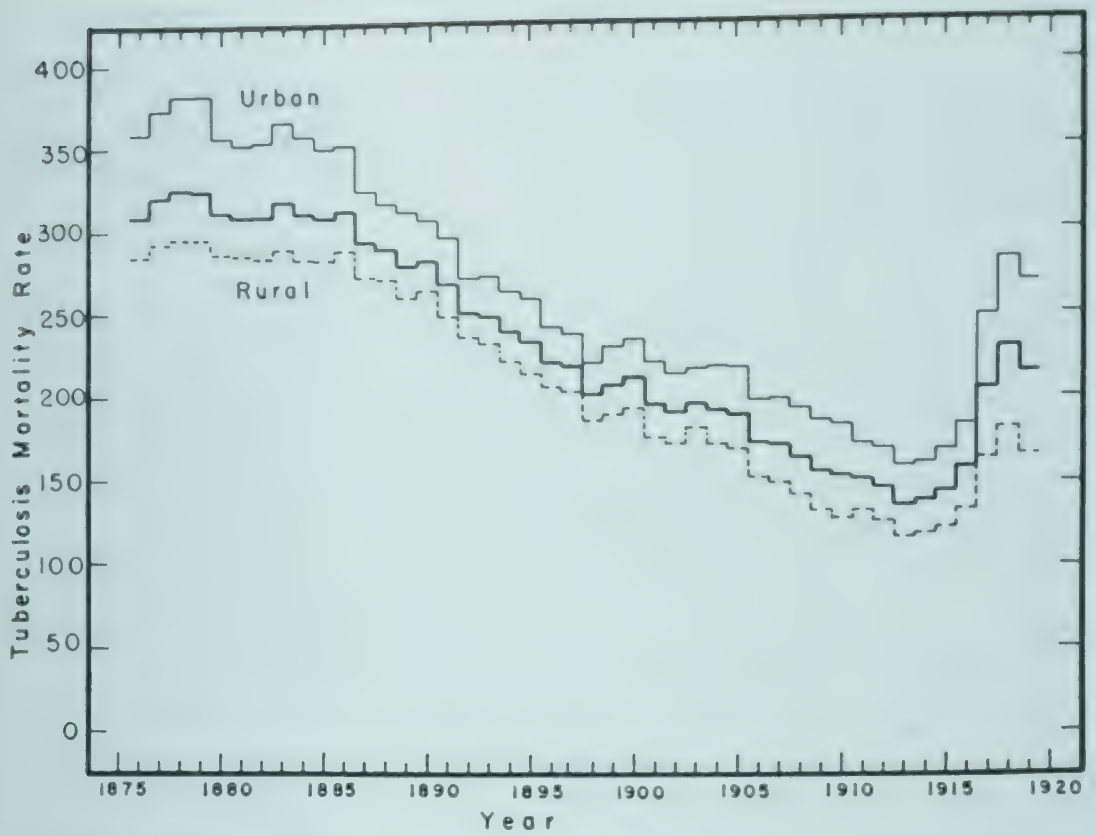


FIGURE 139. TUBERCULOSIS MORTALITY RATE, as deaths per 100,000, in Prussia for the total population and for the urban and rural elements (data from Kirchner, 1921).

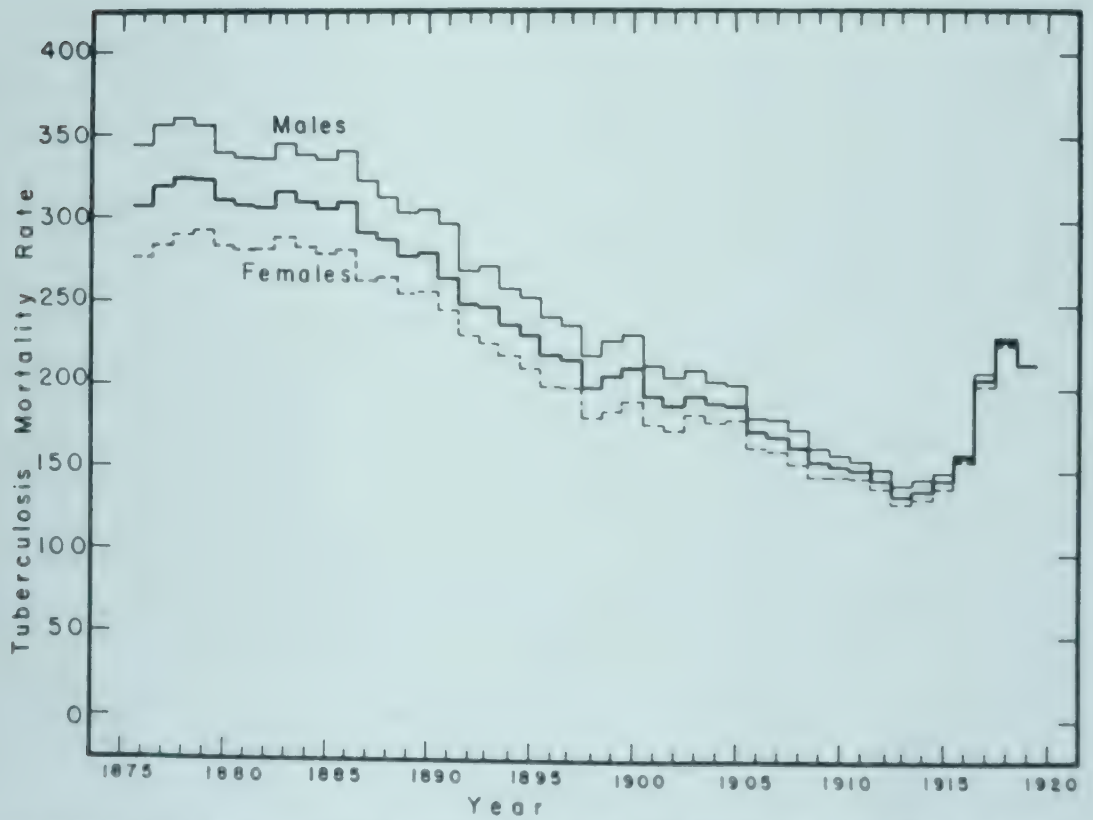


FIGURE 140. TUBERCULOSIS MORTALITY RATE, as deaths per 100,000, in Prussia for males and females and for both sexes combined (data from Kirchner, 1921).

TABLE 422

TUBERCULOSIS MORTALITY RATE IN URBAN AND RURAL AREAS IN GERMANY DURING WORLD WAR I. Rates for each year are given as percentages of the rate for the preceding year and for the year 1913. (Calculated from data given by Kirchner, 1921.)

	1914	1915		1916		1917		1918	
	% 1913	% 1913	% 1914	% 1913	% 1915	% 1913	% 1916	% 1913	% 1917
Urban	101.3	106.9	105.6	116.6	109.0	157.4	135.0	178.7	113.5
Rural	102.0	104.4	102.4	113.9	109.5	141.3	127.4	155.5	110.1

TABLE 423

TUBERCULOSIS MORTALITY IN SAXONY AND IN BAVARIA, the rate per 100,000 of population and as a percentage of the rate in 1914 (Arnould, 1942).

	1914	1915		1916		1917		1918	
	Rate	Rate	% 1914	Rate	% 1914	Rate	% 1914	Rate	% 1914
Saxony	129	129	100	142	110	213	165	257	199
Bavaria ...	174	180	103	195	112	202	116	207	119

except for rather minor and temporary shortages in 1916 and 1917. The deterioration in food supply in the cities was far more serious and prolonged.

The difference is not merely one between larger and smaller communities. Differences in rates of tuberculosis incidence and mortality in Germany during World War I can be shown to be related to local food situations and the productive character of the countryside. At Trier a sharp rise in tuberculosis incidence was coincident with the potato shortage there in 1916-17 (Bruns, 1920). Arnould (1942) pointed out that the wartime rise in tuberculosis mortality in Saxony was far greater than in Bavaria with its much larger food productivity (see Table 423). For Germany as a whole the tuberculosis mortality in 1917 was 44 per cent greater than in 1913, but in relatively well-fed Bavaria the rate rose only 13 per cent at the same time (Drolet, 1945).

A comparison of different cities in Austria indicates further the correlation between tuberculosis mortality and food supply. In Austria the Treaty of Saint-Germain made Vienna effectively dependent for food in 1919 on a forest and mountain hinterland; the food shortage was acute and the mortality rate from tuberculosis rose almost 100 per cent in 1919 as compared with 1913. In contrast, Salzburg, which underwent the same housing shortage and other war-produced stresses, had a plentiful food supply from its more agricultural surroundings, and the tuberculosis mortality rate rose only 15 per cent at this time.

As indicated in Figure 140, the two sexes did not share equally in the tuberculosis mortality in Germany. Before World War I the total male death rate from tuberculosis had exceeded that of the females for many years. This difference steadily decreased during the war until in 1918 the female death rate actually surpassed that for the males. There are two significant points to observe. In the first place, the difference, both absolute and in percentage terms, between the male and female tuberculosis mortality rates had been decreasing steadily since

1887. In the second place, the sex ratio of tuberculosis deaths had always been much more unfavorable to the males in the cities than in the rural areas. For the period 1899 through 1913 the mortality from tuberculosis in the urban population averaged 24 per cent greater for males than for females; in rural areas the difference was only 2.3 per cent. For the war years the pertinent data are summarized in Table 424.

The female tuberculosis mortality rate in the cities showed the greatest relative rise, but in both urban and rural areas it is impossible to estimate the role of nutrition. In the country the women did much more of the farm work than before, but they had fairly adequate food supplies. In the cities the women left their homes for all sorts of clerical and industrial work. Both men and women migrated to the cities in substantial numbers, and of course large numbers of men were absorbed by the army, so that the several population groups are not entirely comparable. At most it might be suggested that the food shortage in the cities worked a special handicap on women, who were doing far more work and were exposed to many more personal contacts than they were accustomed to. It may be noted that in the Netherlands and in Switzerland the rise in wartime mortality from tuberculosis was more marked for women than for men, but the opposite was true in Denmark.

TABLE 424

TUBERCULOSIS MORTALITY IN GERMANY IN WORLD WAR I; males and females in urban and rural populations. Rates are given per 100,000 persons of the same sex and as percentages of the rate in 1914. (Kirchner, 1921.)

Year	Urban Population				Rural Population			
	Males		Females		Males		Females	
	Rate	% 1914	Rate	% 1914	Rate	% 1914	Rate	% 1914
1914	175.8	100	145.1	100	116.2	100	121.3	100
1915	183.2	104	155.4	107	119.3	103	123.9	102
1916	192.3	109	176.7	122	126.5	109	138.3	114
1917	263.1	150	235.6	162	159.5	137	169.5	140
1918	288.5	164	177.1	191	174.6	150	187.6	155

The rise in tuberculosis mortality during World War I did not affect all ages alike. The increase was greatest, both absolutely and relatively, in the age group 15 to 30 years. Least affected were the infants under one year of age. In absolute rates the age group 1 to 15 years showed, next to the infants, the least increase, but the percentages increase was exceeded only by the 15- to 30-year group. Again it is difficult to suggest specific relationships to nutrition except in the case of the infants, who almost certainly received the most adequate food. The mortality rate of tuberculosis in infants less than one year old in Germany actually continued to decrease until 1917 (Kirchner, 1921).

The food situation in Central Europe during World War I has been discussed many times. Here it is enough to say that there was no serious undernutrition, or at least no real change in the general nutritional level, until 1915, and, though bread, potatoes, and pork had to be rationed, the food situation was not

bad until 1916; crop failures in that year resulted in widespread undernutrition which gradually became worse in 1917 and 1918.

It is easy to draw a close parallel between German food consumption and the increase in tuberculosis. But, of course, almost exact parallels could also be drawn for such items as the long working hours, increased personal contacts, housing congestion, and so on. Open cases of tuberculosis were often no longer isolated and were even considered employable in many instances. Kirchner (1921) stated that in Berlin there was at least one tuberculous person sharing a room with others in 49 per cent of all residence buildings (mostly apartment houses). All these factors were certainly contributory. But it is significant that almost all investigators on the scene were positive that undernutrition was by far the most important factor in lowering the resistance of the population to such a point that tuberculosis became a rapidly fatal and epidemic disease (Anon., 1924; Kirchner, 1921).

Denmark—World War I

One of the best investigations on the relationship of undernutrition to tuberculosis mortality is the well-known study of Faber (1938) in Denmark. Only the most pertinent facts need discussion here.

As in Germany, tuberculosis in Denmark had been declining for many years before 1914. In World War I the urban mortality rose rapidly to a peak in 1917, one year before the maximum in other European nations, and declined thereafter, with a slight break in 1924, to regain the level which would have been predicted, in the absence of war, from the long-time trend. The data for 1913–22 are summarized in Table 425.

The tuberculosis mortality data for Denmark, and particularly the deviations from those of other European countries, offer the strongest argument for the great importance of the factor of nutrition. Before the war Denmark exported large quantities of bacon, eggs, and dairy products and imported grains, animal feedstuffs, fertilizer, and fuel. During 1914 and 1915 this trade continued under agreements with the several belligerents. The exports increased but the imports,

TABLE 425

MORTALITY FROM ALL FORMS OF TUBERCULOSIS IN THE URBAN POPULATION OF DENMARK AND IN THE TOTAL POPULATIONS OF 4 OTHER COUNTRIES. Rates are given per 100,000 of population and as percentages of the rates in 1913. (Faber, 1938.)

Year	Germany		Italy		England		Holland		Denmark	
	Rate	% 1913	Rate	% 1913	Rate	% 1913	Rate	% 1913	Rate	% 1913
1913	142	100	156	100	134	100	141	100	135	100
1914	143	101	145	93	136	102	140	99	138	102
1915	148	104	157	101	147	110	144	102	132	98
1916	162	114	166	106	146	109	168	119	155	115
1917	206	145	175	112	151	113	181	128	176	130
1918	230	162	209	134	157	117	203	144	138	102
1919	211	149	172	110	126	94	173	123	119	88
1920	154	108	160	103	113	84	147	104	117	87
1921	137	97	142	91	113	84	127	90	107	79
1922	142	100	143	92	112	84	115	82	101	95

particularly of rye and wheat, declined, and Denmark's creditor position increased. Domestic prices rose. In 1916 shipping difficulties began but food prices continued to rise beyond the purchasing power of the lower income groups. In February 1917 the German unrestricted submarine warfare and the Allied counterblockade suddenly reduced all shipping to a trickle, and the imports of animal feedstuffs declined to about a third of the 1913 level, while bacon and butter exports decreased by 21 and 36 per cent, respectively. The domestic food supply increased sharply, not only because of the reduced exports but even more because it was necessary to slaughter great numbers of hogs and cattle. Rationing and government subsidies secured fairly even distribution, so that the nutritional state of all the urban population was greatly improved.

Faber emphasized the fact that the domestic food consumption, particularly of proteins, was closely related (inversely) to the tuberculosis mortality, but that other factors which might also affect the mortality were not coincident in time. For example, housing was poor, but the worst overcrowding occurred after the war, when the tuberculosis mortality was back to the prewar trend. Fuel was scarce in 1917 and continued so in 1918. Unemployment reached a low point (4.9 per cent) in 1916 and a maximum (18.1 per cent) in 1918. Medical and public health facilities were not adversely affected except by the influenza epidemic of 1918 (4150 deaths in a mortality of 18,000 from all causes).

England—World War I

The mortality rate for tuberculosis in England rose during World War I (cf. Table 425), and the changes were more moderate than in Germany. Both these facts might seem to be in line with the nutritional and general conditions, but Cobbett (1930) examined the detailed statistics and arrived at conclusions rather different from first expectations. He argued that the hastened death of persons already tuberculous would account for most of the increase apparent in 1915 and that the influenza epidemic in 1918 hastened the death of many patients and gave a fictitious peak in that year. He considered the prompt fall after the war to be due to the prior death of most of those with severe infections rather than to the improvement in nutrition and living conditions.

Cobbett also objected to the figures usually cited for England, which are calculated on the civilian rather than the total population. When the latter basis is used, the wartime rise in the tuberculosis mortality for males is much less marked. Finally, Cobbett pointed out (p. 101) that the calculated excess of tuberculosis deaths, over the 1914 figure, for the four years 1915–18 amounted to 20,998 for all of England and Wales. But 5147 of these excess deaths were contributed by the mental hospitals, whose population represented only some 0.2 per cent of the entire country. Health conditions were bad in these institutions, and there were enormous increases in other diseases, such as dysentery.

In effect, Cobbett concluded that the rise in tuberculosis mortality in England was not great, that changes in the time of death of patients already infected were involved, and that tuberculosis morbidity was not much increased by wartime conditions, including food shortages. He cited the siege of Paris in 1870–71, when tuberculosis mortality rose 35 per cent in five months but there-

after fell below all previously recorded levels and only regained the general trend line in 1880. This phenomenon might be explained as due to the death during the siege of tuberculous persons who otherwise would have contributed to the mortality data for 1872 to 1879.

It is true that tuberculosis patients do not fare well in wartime and that they adapt poorly to the less palatable diet even when it is offered in caloric sufficiency (cf. Keers, 1945). A commission appointed to examine into the high tuberculosis mortality in the English mental hospitals during the war found both the quantity and the quality of the food to have been reduced and concluded that therein lay the main reason for the increased mortality.

In the years 1914–18 there were food shortages in England, particularly of meat and dairy products, but serious caloric undernutrition was certainly not general at any time, and it is not possible to place the major blame on nutrition for the moderate increase in tuberculosis mortality in the population at large.

World War II—The General Picture

The data on tuberculosis during and after World War II are still fragmentary and, such as they are, need further analysis before they can be accepted as final. The general situation differed from that in World I because of the more widespread disruption and overcrowding caused by bombings, the far greater mass movements of people, including the “slave laborers” impressed into service in the enlarged German Reich, and the Nazi policies of studied neglect of non-Nazis and actual extermination of anti-Nazis. At the same time the distribution and rationing of the food supplies was better than in World War I.

Tuberculosis increased everywhere, but there were great variations between regions and, particularly in Nazi-controlled areas, between elements of the population. The conditions in prison and concentration camps ranged from bad to incredibly frightful. Markowski (1945) estimated that in the New Brandenburg Camp for prisoners of war in 1944 there were 2000 cases of active tuberculosis out of a total of 6000 prisoners. Conditions were as bad or worse in German concentration camps for political prisoners. Nearly a third of the Netherlands deported for slave labor by the Germans returned with tuberculosis (Kars, 1946). Debray *et al.* (1946) examined 771 men, mostly political prisoners, removed from the infamous German concentration camps; 400 of them were tuberculous. In spite of hospital care, there was a total of 67 deaths, 35 from tuberculosis, 3 from exanthematous typhus, and 29 from extreme cachexia and diarrhea; most of this last group died within a few hours after arrival at the hospital. Professor Richet estimated that 40 per cent of the deaths at Buchenwald were the result of tuberculosis (Lamy *et al.*, 1948, p. 223).

In World War II the ascertainment of tuberculosis was improved in some countries, notably in Great Britain, the United States, and other countries where greater authority of the central government was coupled with a keen sense of the importance of tuberculosis control. In such areas the World War II mortality data probably have a small upward bias as compared with the preceding period. In these countries the introduction of mass chest X-rays produced so large a bias in tuberculosis morbidity, compared with prewar incidence, that no accurate

comparison is possible. In some other countries, such as France and Greece, the general chaos probably resulted in a downward bias in ascertainment of tuberculosis morbidity and mortality. The mortality statistics for these areas show large percentages of the total deaths listed as "cause unknown" or "no medical certificate" or "not classified."

The net result is that the World War II data must be used with great care in attempting to discern relationships between diet and tuberculosis. But in spite of all these limitations, the information for the years 1939 through 1946 provides much valid confirmation for the belief that caloric undernutrition exerts a highly unfavorable action on the course, and probably the incidence, of tuberculosis.

Germany and Austria—World War II

At the time of writing, information about tuberculosis in Central Europe in World War II is still fragmentary and based on unsatisfactory data. The fact that in many regions the food situation became very bad only near the end of the war and immediately after its close means that the nutritional effects were inextricably confused with those attending the final physical warfare, the complete collapse of civil authority including public health control, mass migrations and evacuations, and the division of the countries into different zones of occupation.

Tuberculosis increased in Germany during World War II, the most serious change being in the eastern part of the country (Stowman, 1946b). In Austria the situation was similar. In Vienna the mortality from tuberculosis averaged 114 per 100,000 inhabitants in 1937–39; in 1943 the rate was 135, and in 1945 it rose to 251. The data from Vienna for the first two months of 1946 indicated an annual rate of about 300 per 100,000, compared with first quarter rates of 236 for 1945 and 125 for 1937–39 (Stowman, 1946a). The changes after the conclusion of hostilities, particularly for 1946, coincided with a further deterioration in the nutritional state even though there was a restoration of order.

England—World War II

The experience of England in World War I prompted much greater attention in that country to nutrition in World War II. The program of the Ministry of Food laid stress on the importance of "protective" foods, particularly the production of vegetables and milk, and paid attention to the food provided in the institutions where neglect had prevailed in the previous war. Nevertheless, deaths from tuberculosis in England rose promptly, a small increase occurring even in 1939, when only four months of war had certainly not affected the national nutrition. In 1940 the deaths from tuberculosis in England and Wales totaled 28,151, compared with 25,589 in 1938; in 1941 the total was 28,667 (Anon., 1942). In spite of the efforts to aid the mental hospitals, the tuberculosis mortality for 1941 in 10 of the larger institutions had increased, in comparison with 1937–39, by 70 per cent for males and 10 per cent for females. In subsequent years the food shortage, never very acute, was scarcely improved, though food distribution methods became more efficient. But tuberculosis mortality steadily declined, reaching a record low of 58.3 per 100,000 in 1944 (Magee,

1946). The incidence of tuberculosis was elevated above the immediate prewar figure—54,300 in 1943 and 50,689 in 1938—but this may be illusory. As Magee (1946) pointed out, the increase was “unquestionably, to some extent, a measure of improvement in ascertainment rather than in real incidence” (p. 10).

It was the opinion of the British Medical Research Council in 1942 that malnutrition was not important in the rise in tuberculosis in the first years of the war (Anon., 1942). Other authorities concurred in this view (MacNalty, 1942; MacKenzie, 1943; Westwater, 1945). Keers (1945) noted an increase in new patients entering a sanatorium catering to the wealthy; he cited this as an example of increased tuberculosis without any factor of undernutrition.

In Scotland tuberculosis rose rather sharply in World War II. In 1943 there were 54 per cent more new cases than in 1938, which Westwater (1945) attributed in large part to the deplorable housing conditions. Laidlaw and MacFarland (1942), in explaining the rise in tuberculosis in Glasgow, considered malnutrition relatively unimportant compared with increased personal contacts, poor housing, and overwork.

In short, the situation in Great Britain in World War II was that tuberculosis morbidity rose slightly and mortality increased only in the first half of the war. There was a chronic food shortage but little or no real undernutrition and perhaps actual qualitative improvement in the average diet. Magee (1946, p. 10) stated there is “good reason to believe that the well-balanced diet during the war did a great deal to prevent increased mortality from tuberculosis.” Such changes in tuberculosis as did occur are explicable by the closing of sanatoriums, poor housing, increased industrialization, and chronic fatigue (MacKenzie, 1943).

France—World War II

French mortality and morbidity figures are open to some question because tuberculosis is not a reportable disease, and, except in Paris, deaths are not necessarily certified by a physician. But there are useful data from World War II, particularly from the Department of the Seine, where there was a progressive fall in tuberculosis mortality from 1930 to 1939. An increase was recorded in 1940, and in 1941 the level was 36 per cent above that for 1939 (Anon., 1945d). These changes agree in general with those from other sources for France as a whole (Besançon, 1945).

There were wide variations in tuberculosis mortality in different parts of France. The rate in Brittany was lower in 1943 than in 1938, while the industrial areas of southern France suffered great increases. Although population shifts and large numbers of deaths recorded as from unknown causes or not medically certified complicated the picture, there seems to be a clear relationship between the mortality rate in different parts of France and the local nutritional situation. There was little or no malnutrition in Brittany, but undernutrition was very serious in the industrial south (Besançon, 1945; Magee, 1946). Rates for 1938 and 1943 are summarized for 8 French departments in Table 426.

Clinically, three stages were distinguished in tuberculosis in France (Ameuille, Fauvet, and Renardt, 1944): (1) from September 1939 to the winter of 1940–41 there was little increase in tuberculosis and no change in its clinical

TABLE 426

MORTALITY FROM TUBERCULOSIS PER 100,000 INHABITANTS IN DIFFERENT DEPARTMENTS OF BRITTANY AND SOUTHERN FRANCE IN 1938 AND 1943 (data from Epidemic Information Bulletins, 1945).

Department	1938	1943	
	Rate	Rate	% 1938
Côtes du Nord	257	180	70
Finistère	306	174	57
Loire-Inférieure	243*	150	62
Morbihan	307	148	61
M for Brittany	278	163	63
Rhône	149	165	111
Bouches-du-Rhône	133	231	174
Var	131	221	169
Vaucluse	130	178	137
M for southern France	136	191	148

* Data for 1936.

character; (2) in the period 1941-43 there was a marked increase in both morbidity and mortality, with many more cases of rapidly fatal types; (3) from 1943 to 1944 there was a gradual decrease in morbidity and mortality, with relatively more chronic forms of the disease.

There seems to be reasonable agreement that the foregoing was the clinical picture and that nutritional factors were importantly involved (Ravina *et al.*, 1941; Arnould, 1942; Forel, 1943; Bourgeois, Vie, and Bellin, 1943; Ameuille, Fauvet, and Renardt, 1944; Vignal, 1945). All the French physicians observed far more cases of tuberculous pneumonia, tuberculous meningitis, caseation and rapid cavitation, miliary spread, tuberculous polyserocites, and rapidly progressive glandular tuberculosis. Many of them had a violent onset almost like typhoid and proceeded to death in 3 months or less (Vignal, 1945). While the French authorities recognized the numerous adverse factors involved, the most important cause for the changed character of the disease was believed to be under-nutrition. Forel (1943) noted the inverse parallel between changes in the food supply and tuberculosis mortality. Bariety (1945) stated that Paris firemen, who underwent no reduced rations, had no increase in tuberculosis. Mental institutions and penitentiaries showed increased tuberculosis with greatly enhanced virulence, and the only real change in living conditions there was in food (Forel, 1943; Gerard, Plancher, and Viallier, 1943). In one large mental hospital in Paris a sharp rise in tuberculosis morbidity and mortality coincided with the greatest food restriction and weight loss (Bourgeois, Vie, and Bellin, 1943).

Data on the grants of long leaves of absence because of tuberculosis were gathered by the French National Institute of Health. The postal, telephone, and telegraph services maintained from 134,380 to 146,885 government employees during the years 1936 through 1943. Tuberculosis leaves of absence ranged from 506 per 100,000 (1936) to 421 (1942), except for 1941 when only 342 such leaves were granted per 100,000 employees (U.N.R.R.A., 1945a, 1945b). The low level in 1941 probably indicates greater stringency in granting leaves rather

than any real change in morbidity, but in general this group, in contrast to the general population, gave no evidence of increased tuberculosis. It should be noted that, on the whole, this group also received better rations than the average citizen. On the other hand, employees of the Finance Department and of the Paris Subways, whose advantage, if any, in rations is less clear, reported about 50 per cent increases in tuberculosis morbidity.

Other Countries—World War II

In Canada there was a rise in tuberculosis mortality in 1940 with no change in the general state of nutrition (Wherrett, 1942). Such facts indicate the caution which must be exercised in attributing all changes in tuberculosis in war-time to undernutrition.

In the Netherlands (Dols and van Arcken, 1946) the mortality rate from tuberculosis had been quite low in the years just preceding the invasion by the Germans in May 1940, being only 41.0 per 100,000 in 1939. There was a slight rise (to 43.7) in 1940, but thereafter the rate rose rapidly, being 59.2, 61.3, and 69.9 in the years 1941, 1942, and 1943, respectively. In the first half of 1944, the last period for which Dols and van Arcken gave data, the rate was 82.8, or almost double that of 1939. During all this time there were constant food shortages but no famine, and the degree of undernutrition was only slight to moderate. The rationing program was complete, effective, and admirably equitable. There was no breakdown in medical or public health services. It should be noted that food shortages in the Netherlands began in the latter part of 1939, owing to the cessation of shipping; the Netherlands normally imported large amounts of cereals and flour as well as animal feedstuffs.

Van Vliet (1947) has made a more detailed analysis of tuberculosis up to 1944 in the Netherlands. At the District Consultation Bureau of Groningen both first and total consultations for tuberculosis remained practically constant from 1938 through 1940, but they progressively increased thereafter. In 1944 there were 2½ times as many consultations as in the years 1938–40, and the rate of new consultations had increased fourfold (see Table 427). But these figures do not give a valid index of tuberculosis morbidity for two reasons: (1) there was increasing fear of tuberculosis among the population, and (2) extra rations were provided to persons judged to have tuberculosis.

The detailed data of the Groningen Bureau provide information on the character of tuberculosis for the period 1938 through 1943. Some of the data are summarized in Table 428. It is instructive to compare the figures for the two years 1942–43 with the two prewar years 1938–39. The active cases analyzed in detail for these periods totaled 1278 and 554, respectively. In the prewar period

TABLE 427

NUMBER OF CONSULTATIONS FOR TUBERCULOSIS IN THE DISTRICT CONSULTATION BUREAU AT GRONINGEN, THE NETHERLANDS, IN THE YEARS 1938–44 (van Vliet, 1947).

	1938	1939	1940	1941	1942	1943	1944
First consultations	1933	2006	2245	2714	3078	4143	8043
Total consultations	8240	8218	8223	9638	10155	15468	21206

TABLE 428

DATA ON ACTIVE TUBERCULOSIS IN GRONINGEN, THE NETHERLANDS, FROM THE RECORDS OF THE DISTRICT CONSULTATION BUREAU for the years 1938-43
(summarized from van Vliet, 1947).

	Number of Cases					
	1938	1939	1940	1941	1942	1943
Total	255	299	317	462	517	761
Males	113	153	161	212	245	359
Females	142	146	156	250	272	402
Less than 15 years of age	56	78	48	84	80	129
15-19 years old	42	52	64	69	71	113
20-29 years old	94	85	109	153	180	256
30-49 years old	46	58	72	126	132	185
Over 50 years old	17	26	24	30	54	75
Urban	102	105	110	145	186	305
Rural	153	194	207	317	331	456
Exudative with cavitation	54	47	75	52	70	106
Exudative without cavitation	21	26	41	43	53	104
Primary infiltration	9	6	4	14	10	41
Hilus gland T.B. and perihilar infiltration	29	74	44	84	88	116
Exudative pleuritis	39	56	70	96	99	184
Adhesive pleuritis (dry)	3	7	3	13	2	14
Erythema nodosum	10	26	25	47	37	78
Bone and joint T.B.	6	11	14	13	24	16
T.B. peritonitis	2	5	4	7	8	13
Miliary pulmonary T.B.	1	2	6	5	1	4
T.B. meningitis	0	0	0	4	2	4
T.B. of neck glands	18	14	13	54	51	53
Died within 6 months	8	3	15	14	11	14
Died within 1 year	6	10	16	7	14	22
Died within 2 years	6	10	14	10	11	13

a positive sputum was recorded in 32 per cent of the active cases; in 1942-43 the percentage was 21.5. The proportion of the active cases with extrapulmonary spread of the disease may be estimated from the sum of the cases of tuberculosis of the eyes, bones and joints, meninges, peritoneum, skin, and urogenital system. On this basis there was no significant difference between 1938-39 and 1942-43, the percentage figures being 14.8 and 17.2, respectively. In the war period, however, there was some decrease in the percentage of active tuberculosis with the exudative form of the disease with cavitation, the respective percentages being 18.2 and 13.8.

The Dutch experience up to the famine of 1944-45 may be summarized as showing substantial and progressive increases in tuberculosis mortality and morbidity, due in some small part to improved ascertainment, beginning some months after the seizure of the country by the Germans. For this period there were no marked changes in the character of the disease. The increased incidence and death rate may be blamed on a continued state of moderate undernutrition and a general deterioration of living conditions, but not to any decline in medical care or public health control.

In the western part of the Netherlands true famine conditions developed toward the end of 1944 and continued until May 1945. Detailed analyses of tuberculosis in this period are not available, but it is known that there was a serious rise in tuberculosis mortality. In Amsterdam, for example, the mean rate for 1938 and 1939 was 38 per 100,000 inhabitants, but in the first half of 1945 the rate was 138 per 100,000 (Stowman, 1945).

In urban Poland there was a rise of about 300 per cent in tuberculosis mortality during the war; in Warsaw in 1944 the tuberculosis death rate was over 500 per 100,000 (Daniels, 1946). Mass X-ray surveys showed an incidence of 12 per cent in railway workers, and among students in Krakow and Lublin the incidence was 11 and 12 per cent, respectively. In the sanatoriums the success of treatment was most discouraging, owing to the shortage of food and doctors; in the big Kamiewiegura Sanatorium the available food averaged only 1995 Cal. daily, including 67 gm. of total proteins.

In Japan tuberculosis increased during World War II, and attempts have been made to ascribe this to malnutrition (U.S. Strategic Bombing Survey, 1947, p. 90). Actually, the available data (*ibid.*) show that tuberculosis began to increase in 1935 and that the change thereafter was slight; in 1935 there were 207 tuberculosis deaths per 100,000, and in 1943, the last year for which data are provided, the rate was 225. The most clear-cut contributory cause of increased tuberculosis in Japan would seem to be the increasing industrialization, with a population shift to densely peopled urban regions, associated with the production of war matériel. It should be noted also that there was certainly much improvement in ascertainment during this same period. There is a good deal of doubt about the actual extent of undernutrition in Japan in World War II; in any case there is no basis for claiming an important change in tuberculosis related to the diet.

In Russia there was a rise in tuberculosis incidence in Leningrad in December 1941 which became progressively more serious until May and June 1942, and there were important changes in the character of the disease (Brožek, Wells, and Keys, 1946). Satisfactory data for analysis are not available, but the general picture was one of a definite parallel between tuberculosis and the degree of malnutrition.

In Belgium the food situation was poor but never very bad during the German occupation of 1940–44. Losses in body weight were of the order of 5 to 10 per cent and occurred mainly in early 1941 and again in the spring of 1944. The mortality rate from tuberculosis rose from 68 per 100,000 in 1939 to 95 in 1943. The reported cases of tuberculosis totaled 31,069 in 1939 and 131,380 in 1943, but there are doubts that these figures are properly comparable.

Greece suffered severely from undernutrition in World War II, and famine conditions prevailed in the latter part of 1941 and most of 1942. Valaoras (1946) estimated a population loss of over 60,000 from excess mortality and decreased natality in Athens and Piraeus (population 957,000 in 1940) during the two worst years. This was a case of "supervised" famine, since the Germans maintained order; there were no epidemics and full records were kept. The data summarized in Table 429 are instructive. Tuberculosis mortality rose somewhat be-

fore the German occupation when food conditions were not very bad. But tuberculosis mortality was more than twice as great in 1942 as in 1940. In the latter half of 1943 and the first half of 1944 the mortality declined sharply. Rather similar changes occurred in mortality from diseases of the digestive, circulatory, nervous, and genitourinary systems. These data suggest that severe undernutrition increases the mortality from many diseases besides tuberculosis. The additional stress of undernutrition may well result in premature death or a fatal outcome to a condition that could otherwise be tolerated.

In 1946 it was said of Greece that "tuberculosis and malaria constitute a far greater menace to life and health than do the epidemic diseases" (Stowman,

TABLE 429

MORTALITY IN ATHENS AND PIRAEUS, BY CAUSES, FOR 1940 THROUGH FEBRUARY 1944. The data for 1943 and 1944 covered only 72 per cent of the population but have been adjusted to the equivalent of 100 per cent in this table. The figures listed are the total deaths and these deaths as percentages of the corresponding figures for 1940. The "all other" category is made up chiefly of deaths from senility, violence, and starvation, the latter being the cause of the bulk of the deaths in this category for 1941 and 1942. (Data from Valaoras, 1946.)

Cause of Death	1940		1941		1942		1943		1944
	Jan.- June	July- Dec.	Jan.- June	July- Dec.	Jan.- June	July- Dec.	Jan.- June	July- Dec.	Jan.- Feb.
Tuberculosis									
Total	1,287	1,244	1,498	1,820	2,588	2,425	2,039	1,535	540
Percentage of 1940...	100	100	116	146	201	195	158	124	
Respiratory diseases, non-T.B.									
Total	907	622	910	1,226	1,681	862	675	400	351
Percentage of 1940...	100	100	100	197	185	139	74	64	
Diseases of the digestive system									
Total	580	892	756	2,240	1,425	1,966	408	803	123
Percentage of 1940...	100	100	130	251	246	220	70	90	
Diseases of the circulatory system									
Total	799	614	822	1,612	1,463	894	832	551	340
Percentage of 1940...	100	100	103	263	183	146	104	90	
Diseases of the nervous system									
Total	683	568	720	958	848	548	425	394	174
Percentage of 1940...	100	100	105	169	124	96	62	69	
Cancer									
Total	490	500	424	540	359	359	337	483	124
Percentage of 1940...	100	100	86	108	73	72	69	97	
Genitourinary diseases									
Total	434	391	438	964	868	604	372	298	106
Percentage of 1940...	100	100	101	247	200	154	86	76	
All other causes									
Total	1,700	1,637	2,348	8,920	17,692	6,229	2,272	1,992	1,098
Percentage of 1940...	100	100	138	545	1,044	380	186	169	
Deaths from all causes									
Total	6,880	6,468	7,916	18,280	26,924	13,887	7,360	6,456	2,856
Percentage of 1940...	100	100	115	283	391	215	107	100	

1946c). Similarly serious postwar tuberculosis problems were reported for Poland and Yugoslavia, where severe undernutrition was likewise present (Stowman, 1946b).

Italy suffered a moderate rise in tuberculosis mortality early in the war (80 per 100,000 in 1939, 102 in 1942) which does not seem to have been related to the nutritional state; U.N.R.R.A. (1945) cited overcrowding, breakdown of sanitary control, and the closing of sanatoriums as important contributory causes. Since 1942 the data are incomplete but are reasonably good for Rome, where civil disorder and the physical impact of the war were at a minimum. In Rome the tuberculosis mortality rate was 118 in 1939 and 243 in 1944. The rate began to fall at the end of 1944 and decreased further in 1945, though remaining far above the prewar level (Stowman, 1946c). The parallel between tuberculosis mortality and the nutritional conditions in Rome seems to be very close.

All countries of Europe suffered some stresses of war—rationing programs, disordered economies, excessive military establishments, qualitative changes in the available foods, and so on. It is interesting to note that in general the neutral countries (Sweden, Spain, Switzerland, and Portugal), where no special undernutrition developed, showed no changes in tuberculosis (U.N.R.R.A., 1945b). But Ireland, which underwent small but appreciable changes in food supply as well as increased industrialization, showed a sizable increase in tuberculosis mortality (115 per 100,000 in 1937–39, 125 in 1940–41, 145 in 1942–43). Oslo, where food, fuel, and clothing were short but other changes were minimal, had no increase in tuberculosis mortality. Copenhagen, where fuel and clothing were short but food was relatively abundant, likewise had no rise in tuberculosis mortality (*ibid.*).

Even in remote areas where the physical impact of the war was slight and housing and personal contacts changed little, there were changes in tuberculosis mortality wherever there were food shortages and increased work. For example, in the Belgian Congo, van Hoof (1946) stated: "The excess of work and the scarcity of food would necessarily favor this bacillary endemic during the war. As against 877 cases found in 1940 and 764 in 1941, there were 1,003 cases in 1942, 1,194 in 1943 and 1,130 in 1944" (p. 463).

Released Prisoners in Switzerland

The Swiss military hospital at Herisau counted 117 active cases of tuberculosis in a total of 296 patients freed from German prison camps. Labhart (1948) made an extended analysis of the disease in these and 63 similar patients at Davos. These 180 patients with active tuberculosis were rather heterogeneous. Some of them, especially among those evacuated directly from the concentration camps, were extremely emaciated and often totally prostrated, while a few presented a fair nutritional picture by the standards of the camps. At X-ray the great majority exhibited reduced heart sizes similar to those of other starvation victims. There were 58 classified as *primary* tuberculosis and 122 *post-primary*.

The *primary* cases were exudative at first, mostly open T.B., and included 25 patients who showed cloudy to homogeneous shadows or single foci, partly confluent with infiltration, 11 patients who had uniformly disseminated foci, 4 pa-

tients with dissemination and infiltration but with closed lesions, and 18 patients with pleuritis and hematogenous distribution or important lung findings. The criteria which were relied upon for the diagnosis of primary tuberculosis were: (1) lack of calcifications and other residues of tuberculosis in the lungs and hilus; (2) enlarged, poorly defined hilus; (3) bipolarity, rarely seen in the ordinary films but more often clear in the tomoroentgenogram; (4) lack of indications of tuberculosis in the earlier history of the patient or his family; (5) characteristics of the clinical development; and (6) the youthful age of the patients.

Among the 122 cases of *post-primary* tuberculosis, there were 30 patients, mostly with open pulmonary tuberculosis, who had the infiltrated form of the disease, initially exudative, 41 patients with closed tuberculosis, 23 patients with the disseminated exudative form of the disease, 3 patients with hilar findings but questionable distribution and activity, 19 patients with pleuritis and disseminated lesions, and 5 patients with extrapulmonary tuberculosis and no signs of activity in the lungs.

All but 3 of these patients received only conservative treatment for the first 3 to 6 months after liberation from the camps. Subsequently, collapse therapy was used when it was indicated, but Labhart's analysis is limited to the first 8 months of total treatment, so the ultimate use and outcome of active therapy are not indicated. Within the limitations of the total time of observation the over-all results were surprisingly good. Ten of the patients with primary tuberculosis, including 2 with tubercular meningitis, died, but 6 of these deaths occurred shortly after arrival. Five of the patients with the post-primary disease died quickly; there were no later deaths in this group, although 6 of these patients were deteriorating when last seen.

In January 1946, some 8 months after hospitalization, 53 patients in the primary infection group had gained an average of 13.3 kg. (29.3 lbs.) and 77 per cent of them were clinically improving. In the post-primary group at the same time, 82 patients had gained an average of 10.2 kg. (22.5 lbs.) and 86 per cent of them were improving. At that time the sputum was negative in 51 and 59 per cent, respectively, of the two groups, and a few months later the sputum was negative in 6 out of 11 patients who were positive in January 1946.

The surprising course of the disease makes it important to examine as closely as possible the extent and severity of the disease process as presented for treatment. In Labhart's series of 180 patients, there was bilateral pulmonary tuberculosis in 73 per cent and pleuritis as a complication in 24 per cent. Cavernous pulmonary tuberculosis was diagnosed in 54 per cent and 13 patients had bilateral cavitation. Especially among those with primary infections there were frequent instances of a strongly exudative, infiltrating tuberculosis with involvement of the tracheobronchial lymph nodes and disseminated foci. But in the entire series extrapulmonary tuberculosis was relatively rare, and there were no cases of miliary tuberculosis. By and large, the picture is one of relatively severe, rapidly developing activity of recent origin.

Labhart thought that in the majority of cases in his post-primary group the active process was a new infection rather than a reactivation of the older infection. This would be in conformity with the findings of Kars (1946), who investi-

gated repatriated internees in German concentration camps and concluded that starvation and the bad conditions of the camps did not tend to reactivate arrested tuberculosis. Among the 1200 deported persons 29 were known to have had arrested tuberculosis before deportation but only 2 died and the others returned sound, while among the healthy deportees 31 per cent returned with tuberculosis. The data actually suggest that reactivation of fully arrested tuberculosis is unusually infrequent under these conditions. Some such explanation might be offered for the high proportion of primary infections in Labhart's patients.

Labhart believed the character of the disease in his patients to be conditioned by three factors: (1) extremely intense exposure to tuberculosis resulting from the situation in the German camps, where numerous persons with active tuberculosis lived with the rest of the prisoners in incredibly crowded and dirty barracks; (2) severe malnutrition, both qualitative and quantitative; and (3) chronic overexertion and strain. A direct effect of undernutrition on susceptibility was discounted. In support of this view may be cited the remarkably low incidence of tuberculosis among the badly undernourished children studied in East Prussian villages by Ickert (1930). That good nutrition is no protection against tuberculosis is indicated by the observation that well-nourished Danish police assigned to work in concentration camps where there was much tuberculosis rapidly contracted the disease (Cochrane, 1945). Among 350 of these Danes, who received weekly food supplements from Denmark, there were, within 3 months, 7 cases of open pulmonary tuberculosis and 4 cases of pleuritis.

There is a marked contrast between these views and the opinions expressed by some tuberculosis specialists who examined the situation in France and Belgium. It is of interest that Labhart, who obviously was well acquainted with the literature, did not refer to Faber (1938), whose analysis has been generally viewed as perhaps the best argument for the effect of diet on susceptibility to tuberculosis. Reconciliation of the conflicting evidence and opinions, however, does not seem impossible. That wartime conditions, in and out of prison camps, are conducive to increased exposure to infection will be denied by none. Furthermore, there is no dissent from the conclusion that continued undernutrition is highly favorable to the progress of the established infection. Neither of these conclusions is in conflict with the idea that the undernourished tubercular patient may respond well to treatment if he is given a good diet. It is even reasonable to suggest that the alteration in the patient's status on a good diet may be greater when the previous diet has been particularly deficient.

General Evidence on Diet and Tuberculosis

The argument for a general relationship between diet and morbidity or mortality from tuberculosis has been presented many times and need not be reviewed in detail here. Tuberculosis is always most prominent among the poorer people in urban areas; these people tend to be undernourished or at least malnourished in comparison with the rest of the community or with the rural population. Such statistical parallelism does not, of course, establish a cause and effect relationship, and there are many individual exceptions. Moreover, tuberculosis

tends to produce anorexia, so the appearance or recent dietary history of the patient when the disease is recognized may reflect a result rather than a cause of the disease. This must be borne in mind in evaluating such findings as were reported, for example, by Gordon and Flanders (1931). They investigated the diets of patients admitted to a large sanatorium in New York. Immediately prior to their admission these patients consumed, on the average, only about 1400 Cal. daily, and some adults had been subsisting on as little as 800 to 1000 Cal. Potatoes and canned vegetables provided the greatest bulk in most cases, and fresh milk and butter were almost universally absent. But these patients showed no signs of clinical vitamin deficiency.

The tuberculosis situation in the Norwegian Military Training School at Trondheim provided an instructive history (see Leitch, 1945). The tuberculosis morbidity at this school steadily increased until 1921, when nearly one third of the trainees were forced to leave because of tuberculosis. The morbidity rate was many times higher than that of the country as a whole, though the trainees were carefully selected from the standpoint of health. Housing conditions were very poor until 1911, when the school was moved into new modern quarters — with no improvement in the tuberculosis incidence. In 1925 the diet, which had consisted mainly of potatoes, bread, and canned or dried meats and vegetables, was supplemented with fresh milk, fresh meat, cod liver oil, margarine, and fresh vegetables and fruits. The tuberculosis morbidity rate promptly dropped to less than that for the country as a whole. In this case the caloric content of the diet was not changed and only its qualitative composition was altered. Evidence such as this has led to the belief that nutritional quality may be at least as important as quantity.

Dietary Protein and Tuberculosis

Kirchner (1921) in Germany, Faber (1938) in Denmark and Greenland, Flandin and Flandin (1941) and others in France, all have emphasized the inverse correlation between tuberculosis and the consumption of meat and milk. Certainly the impression is current that proteins, particularly those of animal origin, are important in combating the disease. Cannon (1943) has suggested a special importance of protein nutrition in tuberculosis, largely from the Danish evidence. In our general discussion of infections (Chapter 46) we have considered the effect of low protein diets on antibody titer in animals and the relationship to the gamma globulin fraction of the plasma proteins.

High protein diets have often been used in the treatment of tuberculosis (cf. e.g. Pottenger and Pottenger, 1946). The great individual variability in the course of tuberculosis has made evaluation difficult, but at most the benefit achieved has been questionable. The problem has been attacked from another aspect. If it were possible to show that protein metabolism in persons with tuberculosis was qualitatively or quantitatively abnormal, it would provide some reasonable theoretical basis for special attention to protein nutrition in these patients.

McCann (1922) investigated protein metabolism in persons with tuberculosis and concluded that a positive nitrogen balance could be obtained at about

the same level of dietary protein intake as in normal persons or in convalescents. Occasionally, however, unusually high intakes were necessary to cause nitrogen retention. The inference has been made that subacute infections generally, including tuberculosis, produce nitrogen waste or depress nitrogen storage. Johnston and Maroney (1938) studied children before and after the removal of infected tonsils. Following the operation there was a period of considerable nitrogen retention, although the diet was unchanged, and it was inferred that the tonsillar infection had depressed nitrogen storage.

Johnston (1940) noted that in advancing tuberculosis a negative nitrogen balance is common and thought that anything which might promote the nitrogen loss would accelerate the development of the disease. He observed a depression of nitrogen retention in normal girls near the menarche and attempted to correlate this with the increased incidence and virulence of tuberculosis at this age.

A serious objection to much of the work on nitrogen balance in tuberculosis is the fact that it has not taken into account the effect of bed rest alone on nitrogen balance. In the Laboratory of Physiological Hygiene it has repeatedly been shown that bed rest alone will cause a significant negative nitrogen balance in the normal adult, even when the caloric intake is adequate and the protein intake reasonably high (Miller *et al.*, 1945). A positive nitrogen balance is obtained in bed rest only when the protein intake is unusually high. These facts have been confirmed elsewhere (see Chapter 20) and would seem to explain some of the observations offered as proof of an unusual protein requirement in tuberculosis. One of Johnston's (1940) best cases, that of Josephine L., had been studied several times and had shown positive nitrogen balances. Later, however, active tuberculosis was demonstrated at X-ray examination and her nitrogen balance was negative. The exact conditions and time relationships are not stated, but presumably the changed nitrogen balance coincided with the institution of the usual first step in treatment — that is, bed rest.

The confusion regarding the possible relationship between dietary protein and the resistance to or development of tuberculosis is a reflection of the fact that the only measure of resistance has been the course of development of the disease in the person who has been exposed to the infection. Until better measures are available, or long-time controlled mass trials are instituted, the question will remain unsettled.

The Course and Nature of Tuberculosis in Undernutrition

There seems to be unanimity of opinion that the clinical course of tuberculosis is altered in severe undernutrition. Chortis (1946) studied 108 tuberculous patients with famine edema in Athens during the famine of 1941–42. Hospital therapy was limited because of the nursing shortage, the meager food supply, and the impossibility of instituting pneumothorax in most of the patients. The record was that 90 of these patients died, 1 remained stationary, 6 improved, and 6 eventually achieved apparent cures.

In Greece, as in France, there was frequent miliary spread and tuberculous involvement of the viscera and glands, particularly in the thyroid, pancreas, and pituitary glands, where tubercles ordinarily are rarely seen. Small tubercles were

seen in the connective tissue of the heart, spleen, and liver. In 96 per cent of the patients studied by Chortis the pleural cavities showed large collections of fluid, and in over half the pericardium was filled with fluid. In 58 per cent there were ulcerated nodules in the intestinal mucosa, usually in the small intestine, and these were interpreted by Chortis to be tuberculous in nature.

The Greek patients were much troubled by coughing, which was abundantly productive, and hemoptysis was common and difficult to control. The temperature was elevated to as much as 103° F., especially in the evening hours, and there was generally tachycardia. Chortis pointed out the difference from non-tuberculous cases of famine edema and attributed the hyperpyrexia and tachycardia to the toxemia of rapidly progressive tuberculosis.

Larson (1946) reported a detailed analysis of 87 autopsies on tuberculosis victims among Russians and Italians liberated from German prison camps in Italy in December 1944 and January 1945. The average duration of illness before death was recorded as 11.6 months, but Larson stated that this was a considerable underestimate because the Germans made no attempt at early diagnosis. The victims' diet had been extremely bad and, for the 3 months preceding rescue by the Americans, was estimated to have averaged only about 1000 Cal. daily. The average height was 168 cm. (67 in.) and the average weight was 40.8 kg. (106 lbs.). Generalized muscular atrophy was recorded in 91.9 per cent of these men, and 82.8 per cent of them were judged, on the basis of "complete absence of subcutaneous fat," to be severely emaciated. All these bodies showed tuberculosis of one or both lungs. The involvement of other organs is summarized in Table 430.

Larson stated that "evidences of chronic vitamin deficiency were observed in many of those who died soon after admission" (1946, p. 250). The supporting evidence cited, though given only in very slight detail, would make it appear, however, that what were taken to be evidences of vitamin deficiency were, in fact, nothing but the usual results of severe caloric undernutrition. There was generalized edema in 24 of the 87; in only 4 of these were there cardiac or renal

TABLE 430

PERCENTAGE INCIDENCE OF ORGAN TUBERCULOSIS IN 87 CASES OF PULMONARY TUBERCULOSIS IN SEVERELY UNDERNOURISHED RUSSIANS AND ITALIANS rescued from German prison camps (Larson, 1946).

Organ	Percentage	Organ	Percentage
Lymph nodes, thoracic	90.8	Kidneys (caseous)	6.9
Lymph nodes, abdominal	62.0	Kidneys (focal)	9.2
Intestine	54.0	Kidneys, total	16.1
Peritoneum	18.4	Bones	10.3*
Spleen (caseous)	18.4	Pericardium	8.0
Spleen (focal)	48.3	Skin	5.7
Spleen, total	66.7	Genital	1.1
Liver (caseous)	8.0	Esophagus	1.1
Liver (focal)	65.5	Anus	2.2
Liver, total	73.5	Miliary T.B.	3.4

* Considered to be a serious underestimate because the joints and vertebrae were not thoroughly examined.

lesions of consequence; Larson concluded that the others probably had thiamine deficiency simply because they were edematous! The brownish pigmentation of famine, extreme atrophy of the skin, and follicular hyperkeratosis were attributed to niacin deficiency, and so on. It is interesting to note that the antemortem clinical diagnosis in several victims was Addison's disease, apparently on the basis of emaciation, weakness, and skin pigmentation. Obviously, the attending physicians were not well informed about the effects of nutritional deficiencies.

The diagnosis of tuberculosis in famine victims, however, is not always readily made. In the French military hospital at Mainau, Germany, set up in 1945 to receive persons released from the concentration camps, Lamy, Lamotte, and Lamotte-Barrillon (1948) selected for study cases of uncomplicated starvation without infection, but in a few weeks one of their subjects was dead from tuberculosis and several others showed clear signs of the disease which had been missed at first. In other instances a positive but erroneous diagnosis of tuberculosis may be made because of the tendency of starved persons to develop infarct cavities in the lungs from venous thrombi (Hottinger *et al.*, 1948; Lamy *et al.*, 1948) and to produce non-tubercular pleural effusions (Debray *et al.*, 1946; Uehlinger, 1948; Lamy *et al.*, 1948). In some cases these complications are related to a receding exanthematous typhus infection, and in others the pleural effusions seem to have resulted from pneumococcus or staphylococcus infections. The non-tubercular nature of these effusions may be demonstrated by the subsequent course and tests of the effusion fluid injected into guinea pigs.

The behavior of the sedimentation rate is not markedly altered by the presence of undernutrition, even of severe degree, so this test in famine victims may be given its usual significance. Concentration camp victims with tuberculosis tend to have rates as high as 70 or 80 mm. per hour when first seen. This probably means that in such persons the disease tends to be highly active; receding infections are not apt to be seen unless treatment, including a full diet, is instituted. During treatment and convalescence such patients exhibit sedimentation rates similar to those seen in ordinary cases of tuberculosis (Labhart, 1948).

Serological and skin tests, however, may lose much of their value in famine because of general lack of reactivity in grossly undernourished persons. In the severely starved population of the Warsaw Ghetto in 1941 and 1942 it was observed that positive reactions to the Pirquet and Mantoux tests were extraordinarily rare, and negative reactions were the rule also for persons with all degrees of tuberculosis (Braude-Heller *et al.*, 1946). But when the Mantoux test with a $\frac{1}{1000}$ dilution was negative, it was often shown that a 1 per cent solution elicited a positive response.

In the semi-starved men in a mental hospital in France, tuberculosis morbidity rose rapidly as the undernourished state was maintained (Bachet, 1943). In many cases there was little febrile reaction, but the evolution of the disease was rapid. A relationship was seen between the tendency to general edema and the production of pleural effusions. In 15 tuberculous patients who had no edema, there were only 2 instances of pleural effusion; in 29 tubercular patients with edema there was pleural effusion in 22.

All the available evidence supports the conclusion that continued severe

caloric undernutrition, with or without qualitative dietary deficiency, promotes a rapid, fulminating course of the disease, with unusually frequent and extensive hematogenous spread and very high mortality. But, perhaps by the same token, the prognosis may be surprisingly favorable if a good diet can be provided.

In all forms of modern management of tuberculosis, emphasis is placed on a good and abundant diet; efforts are made to produce a gain in weight even when there is no previous emaciation. During the war most regions with rationing programs attempted to give special consideration to patients with tuberculosis, but the needs were not always fully met even in areas where food shortages were not extreme; the results seem to provide further evidence for the value of what might be called overfeeding. In England from 1943 to 1945 the food shortages were reflected in the tuberculosis sanatoriums in spite of preferential treatment; the patients gained less weight than in prewar years and healing was less satisfactory (Keers, 1948).

Original Information

In the Minnesota Experiment all the volunteers were carefully checked for tuberculosis before admission to the experimental group. The potential danger of tuberculosis for these men was emphasized to the subjects, and frequent chest films and clinical examinations were made to detect any development. In September 1945—that is, near the end of the first 12 weeks of rehabilitation—one of the chest films was suggestive, and further studies quickly resulted in a diagnosis of very early minimal tuberculosis in the apex of one lung. This subject (No. 108) had no history of tuberculosis or exposure to the disease prior to the experiment, and he had had a negative Mantoux test on admission. There was no certain history of exposure during the experiment, but two possibilities, neither providing any very intimate contact, could be made out. This subject had shown no peculiar reaction to the starvation, with the possible exception of a specially obvious development of the famine pigmentation which was exhibited by a number of the men.

After initial hospitalization and consultation with this subject's parents, it was decided to send him to his home state for treatment, and full details were transmitted to a specialist near his home. Contrary to the advice of the Laboratory, he did not enter a sanatorium but remained at home under the supervision of his local physician; treatment consisted of several months of bed rest and good food and then a gradual resumption of ordinary activities. He progressed extremely well and in six months his physician reported no trace of the original lesion. About four months later, however, X-ray examination indicated another lesion, again minimal in character. At last reports this subject was progressing well under conservative management.

It was our fortune to have information at first hand regarding a large number of American soldiers with tuberculosis who had been prisoners of war in Japan and the Philippines. The data in no instance were detailed or even reasonably complete, but the picture was uniformly as follows. These men had been, like their fellows, grossly underfed for years in the prison camps and had received neither diagnosis nor treatment from the Japanese. When they were

rescued, almost all were thin and emaciated but practically none showed gross signs of vitamin deficiencies. Diagnosis of tuberculosis was made within a few weeks of rescue, and thereafter they were kept on bed rest and numerous abundant feedings. Their initial recovery, judged by X-ray and clinical observations, was remarkable. In the United States they continued their excellent progress in the military hospitals. Surprisingly, however, after some months a number of these men had relapses or developed new lesions. Their general condition six months to a year after rescue was not greatly different from that of the ordinary tuberculosis patient in spite of their remarkable early improvement.

Diabetes Mellitus and Undernutrition

THE course and, very probably, the etiology of diabetes mellitus are intimately related to the nutritional state. The nutritional problems involved are complex because different types of nutritional or metabolic derangement, frequently operating at the same time, affect the course of the disease and its complications (cf. Guest, 1946). Attention must be given to caloric intakes, balance between the major nutrients, vitamins, and, probably, the mode and intensity of energy expenditure. All these factors are apt to be involved in the effects of general undernutrition, particularly when this is occasioned by limitations in the food supply to a population.

The evidence for evaluating the relationship of undernutrition to diabetes comes from diverse sources: (1) observations on the nutritional status of diabetic persons; (2) mortality data in times of nutritional stringency; (3) clinical results of diet therapy; and (4) studies on the fundamental nature of diabetes. Interpretations are complicated by differences in the incidence and course of diabetes at different ages and in the two sexes, by general trends over the past three quarters of a century, and by the use of insulin therapy. Obviously, we cannot hope, by nutritional analysis alone, to explain in detail the known changes in diabetes mortality in those areas where records are available. Data on the incidence of diabetes are unsatisfactory at best and are totally unavailable for most areas and periods of critical interest. But certain nutritional factors do clearly emerge against the complicating background. Perhaps the most definite point is a favorable effect from a moderate degree of caloric undernutrition on diabetes mortality. Examination of the evidence will provide details on this point and will bring out other aspects of the question.

Diabetes Therapy

In spite of insulin, diet control is still a major element in the management of diabetics and is likely to remain so. A myriad of "diet cures" have been advanced, such as the "oatmeal cure," the "banana cure," the "milk cure," and so on. Many of these are or were successful in reducing glycosuria in individual patients and even in a substantial proportion of the patients on these regimens. It is the general consensus of opinion that the value of these "cures" lies primarily in the coincidental reduction of calories while still providing a reasonable intake of carbohydrate.

Undernutrition has been used in the treatment of diabetes, at least sporadically, for a very long time. The general value of low nutrition in many diseases,

including diabetes, was vigorously championed by Guelpa (1910). More specific and more influential on diabetic management were such studies as those of Allen (1914) and of Joslin (1916), in which it was shown that a reduction in sugar or carbohydrate intake alone frequently increases acidosis but that a general reduction in calories tends to reduce glycosuria and ketonuria. Clinical experience with simple direct replacement of carbohydrates by fats and proteins was unsatisfactory. In the pre-insulin era the custom developed of prescribing a diet carefully calculated never to exceed the caloric needs of the patient and with very limited amounts of carbohydrate. Sugar was forbidden and frequent fast or "green" days were interposed on which the sole fare was liquids or green vegetables with almost no caloric value. On such a regimen it was possible to maintain many diabetics in precarious control and even occasionally to salvage a case of coma.

The introduction of insulin allowed a much less rigid dietary restriction, but, with the accumulation of experience, there has been less relaxation of the dietary control than had been hoped for at first. More carbohydrate is allowed but caloric restriction is enjoined. With insulin, diabetics can take a far more liberal diet without immediate deterioration, but the long-time course of the disease appears to be unfavorable without some caloric restriction. There are marked differences at different ages. Dietary restrictions alone are least satisfactory with younger persons and with children. But with patients of all ages the physician is properly concerned at least to prevent any tendency to obesity or to remove excess fatness if it exists.

Obesity, Glycosuria, and Diabetes

There is much evidence for a fundamental connection between obesity and diabetes. Kisch (1915) found that 50 per cent of all the obese persons he examined exhibited some degree of glycosuria. John (1929) found abnormally high levels of glucose in the blood in glucose tolerance tests in 65 per cent of obese persons who did not show glycosuria on routine examination. Newburgh (1942) reported that 77 per cent of obese diabetics showed a return of the glucose tolerance to normal when their diet was so reduced as to produce a substantial loss of weight. When obesity is induced in rats by lesions of the hypothalamus, they exhibit a striking glycosuria; if obesity is prevented in these rats by dietary restriction, they do not develop signs of diabetes (Brobeck, Tepperman, and Long, 1943).

Statistical analysis indicates a high frequency of obesity among diabetics (Joslin, Dublin, and Marks, 1936). Handelsman (1944), studying diabetics over 40 years of age, showed that weight loss produced by dietary restriction was an important means of control in obese persons whose diabetes was of recent origin. But patients who remained obese for many years became difficult to treat and lost the ability to regain sugar tolerance with dieting. Newburgh and Conn (1939) inclined to the belief that a tendency to the diabetic type of glucose tolerance curve is normal in obese persons. Such persons may not be diabetics or later develop diabetes, though there seems to be unquestionably an increased tendency in this direction.

It is proper to suggest that obesity, which first may only resemble diabetes superficially in a tendency to produce glycosuria and abnormal sugar tolerance tests, may slowly produce irreversible changes, even though weight reduction may correct the glycosuria temporarily. Allen (1946) supports the general clinical impression that in obese persons in whom glycosuria disappears during dietary reduction, true diabetes is very apt to appear later.

When the obese individual is in the actual process of weight reduction, his metabolism is peculiar in that much of his energy is derived from endogenous fat. When caloric balance is achieved at a new and lower weight plateau, the metabolism returns to the more normal situation in which a larger share of the energy is derived from carbohydrates. At this stage the fault in carbohydrate metabolism, which had not been apparent during the weight loss, may again become evident. Young (1945) compared the metabolism during the process of starvation with that in the animal in which diabetes is induced by pituitary extract. In both cases the diabetes may not be evident as long as endogenous fat supplies most of the energy, and in both cases an increase in food intake may be needed to reveal the latent defect. Mahaux (1946, 1947) and others have concluded that there was a real increase in diabetes morbidity in Germany immediately following World War I, and that this was associated with an increased food intake following a period of undernutrition. Such facts would be consonant with the belief that both the maintained state of obesity and the condition in which new fat is being deposited at a rapid rate are conducive to the development of diabetes.

Diabetes Mortality in Germany in World War I

For many years before World War I diabetes mortality and morbidity data showed a steady rise in Germany as in most other civilized countries. German clinicians were therefore impressed when, early in the war, there were indications of an interruption in this unfavorable progression. Elias and Singer (1918) observed a definite improvement in all types of diabetes over the years 1914-18. Eighty-five per cent of those with mild diabetes improved and even in those with severe diabetes, presumably adolescent and non-obese persons, there was some improvement. However, they noted that the proportion of deaths in coma to all diabetic deaths remained much the same. Elias and Singer had an opportunity to study the effect on glycosuria of varying the proportions of carbohydrate, fat, and protein in the diet of several diabetic patients. They found that in some cases the glycosuria varied as much or more with the protein intake as with the carbohydrate or even the total caloric intake.

Magnus-Levy (1919) reported the total diabetic deaths in Berlin over the period 1911 to 1918. In 1915 there was a 15 per cent decrease, and the decrease became progressively more impressive until in 1918 there were less than half as many diabetic deaths in Berlin as the average for the period 1911 to 1914, inclusive (see Table 431). Although statistical data were not provided, Magnus-Levy said that diabetes in hospital patients generally improved so that more of the diabetics who died actually succumbed to tuberculosis and other diseases. He concluded that the general reduction of caloric intake imposed by the war

TABLE 431

TOTAL DIABETIC DEATHS IN SEVERAL LARGE CITIES FOR THE YEARS 1912-18 AND, AS PERCENTAGES OF 1914, FOR THE YEARS 1915-18 (data from Magnus-Levy, 1919, for Berlin; data on the other cities from Mahaux, 1946).

	1912	1913	1914	1915	1916	1917	1918
Berlin							
Total diabetic deaths			444*	385	332	246	202
Percentage of 1911-14			100	87	75	55	45
Breslau							
Total diabetic deaths	100	100	115	73	76	73	52
Percentage of 1914			100	63	66	63	45
Cologne							
Total diabetic deaths	91	91	120	106	97	78	73
Percentage of 1914			100	88	81	65	61
Frankfurt am Main							
Total diabetic deaths	75	83	97	79	64	51	42
Percentage of 1914			100	81	66	53	43
London							
Total diabetic deaths	449	473	492	511	402	402	358
Percentage of 1914			100	104	82	82	72

* Average for 1911-14.

diet was the most important factor in the improvement. Since the war diet was low in protein, he believed that a high protein diet is harmful. But, in keeping with the prevailing fear of feeding carbohydrates to diabetics, he also suggested that the diet must not be too low in fat. In any case, Magnus-Levy was convinced that overloading the body with foods, leading to obesity, is harmful in some way and may lead to diabetes or aggravate the disease if it is already present.

Falta (1919), Strauss (1920), Maase and Zondek (1920), and others reported similar experiences and observations for Germany in World War I. Rosenfeld (1919) observed that diabetes mortality declined 49 per cent in Breslau but only 31 per cent in Munich; the nutritional situation in Munich was considerably better than in Breslau. Mahaux (1946) assembled mortality figures for diabetes in several German cities from 1912 through 1918; these showed essentially the same picture as those from Berlin. There was a definite drop in diabetes deaths in 1915, and this was progressive through 1918, when the deaths were of the order of half those in the same cities in 1914.

Diabetes in England

Mahaux (1946) also listed the total diabetes deaths in London for the years of World War I. These figures showed a drop, beginning in 1916, similar to but smaller than that in the German cities. This corresponds with the later date of real food shortages in England, where food rationing was adopted in the latter part of 1915; a more restricted ration was imposed earlier in Germany. In England as a whole, including Wales, diabetes mortality gradually rose from 1881 to 1915 and then showed an abrupt decline until after World War I (Young and Russell, 1926).

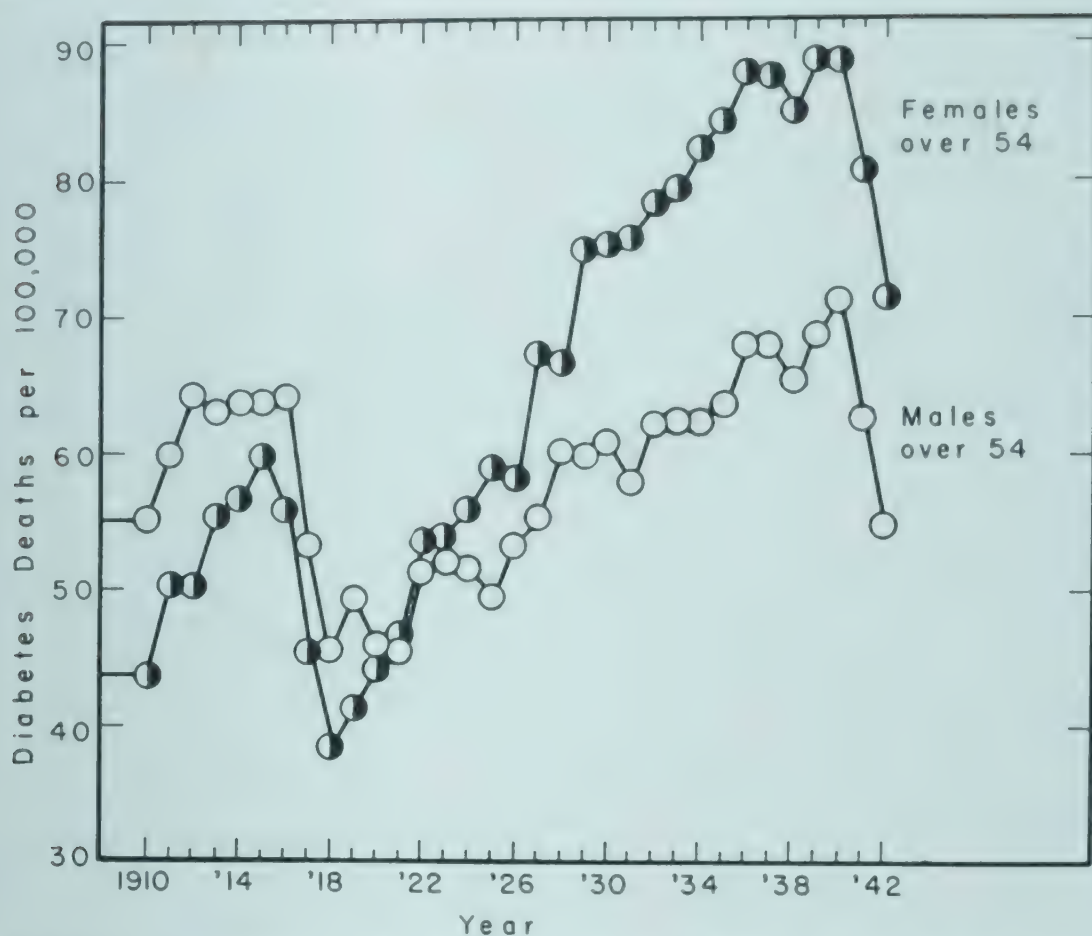


FIGURE 141. MORTALITY RATE FOR DIABETES IN ENGLAND AND WALES, from 1910 through 1942, for older persons (Stocks, 1943).

More recently Stocks (1943) has carefully summarized the statistics on diabetes for all of England and Wales from 1861 through 1942. In general these figures show a steady increase in mortality until the period of World War I. The mortality per 100,000 of population for men and women over 54 years of age since 1908 is shown in Figure 141. This age group is significant not only because diabetes mortality is high in these older people, but more particularly because the complications of military service do not enter. The data show a decline beginning in 1916 for women and in 1917 for men and reaching a minimum for both sexes in 1918, when the mortality was about 30 per cent of the 1913-15 level. If allowance is made for the upward trend before 1916, the decline is significantly greater, especially for women.

The data for England and Wales between the two world wars resemble those of most other countries. The progressive increase in diabetes mortality continued until, for women over 54, the rate in 1940 was $2\frac{1}{2}$ times the 1918 minimum. For men over 54 the rise was considerably less, being, at the peak in 1940, about $1\frac{1}{2}$ times the low of 1918. But with the renewed impact of food shortage and rationing in World War II, there was again an abrupt drop in diabetes mortality in both sexes.

The story is somewhat different for the population under 55 years of age. A

rise in diabetes mortality began after World War I but was abruptly checked in 1923 with the advent of insulin. In the case of the men the mortality rate fairly steadily decreased to reach an all-time low in the late 1930s. In the case of the women a low rate was reached in 1930, which has been maintained with minor fluctuations ever since.

The data for England and Wales clearly bring out one of the most striking characteristics of diabetes statistics the world over. The relative rise in female versus male diabetes mortality is just as impressive in the New World as in the Old (Joslin, Dublin, and Marks, 1936). It is tempting to seek for some explanation of this phenomenon in dietary changes which presumably have affected women more than men. There is no basis for suggesting that women are progressively becoming more obese, or, to put it another way, less undernourished. There is some reason to believe that women have been tending to replace protein with carbohydrate in the diet and, perhaps, to increase their intake of fat.

Diabetes in World War II

We have remarked on the decline in diabetes mortality in England in 1941 and 1942. The data for the remaining years of the war are not, at the time of writing, available to us in the form of satisfactory analyses, but as far as they go they indicate at least a maintained improvement corresponding with the maintenance of a fairly constant level of mild caloric undernutrition. For other parts of Europe, particularly where caloric reductions were severe, the data are relatively fragmentary and are complicated by mass migrations of the populations. However, there are some significant findings.

Diabetic persons in Belgium, France, Germany, and elsewhere were recognized as having particular dietary problems, and official attempts were made to provide for these by supplementary rations. Thus in Belgium the official general ration in 1941 supplied 270 gm. of carbohydrate, 39 gm. of protein, and 19 gm. of fat, making a total of 1440 Cal. Diabetic patients were allowed supplements so that their ration provided 314 gm. of carbohydrate, 68 gm. of protein, and 73 gm. of fat, making a total of 2150 Cal. (Mahaux, 1946, 1947).

Mahaux concluded that this diet resulted in a general improvement of diabetes in Belgium and stated that this would be expected because it was closely similar to the recommendations of Rabinowitch (1930). We must note, however, that the official Belgian ration differed from that of Rabinowitch by providing about 50 per cent more carbohydrate, 30 per cent more fat, and 30 per cent more calories. Moreover, the actual diet in Belgium was not identical with the official allowances. During the winter of 1941-42 butter and meat became almost unobtainable, even with ration cards; potatoes were very scarce, and rutabagas were a mainstay of subsistence. It was believed that later the official ration was more nearly realized. In any case, the general population lost weight, most markedly among the poorer classes. The reduction in food intake was not solely responsible because there was a concomitant increase in physical activity. Owing to the breakdown of public transportation it was necessary to walk or ride bicycles instead of using tramcars and busses. The eternal search for food and clothing required long trips and waiting many hours in line.

In Liège, Belgium, special attention has been paid to diabetes morbidity for some years, and Brull and Decharneux (1943) have analyzed the combined records of clinics, hospital admissions, and outpatient services through 1942. From 1939 to 1942 the total incidence of new cases of diabetes was reduced to about half, and this change was more prominent among females than among males. During the 5 years preceding World War II, 68.5 per cent of the diabetics were females; after 3 years of war this proportion had been reduced to 55 per cent. Brull and Decharneux emphasize three causes: the reduction in overnutrition and establishment of a general state of moderate caloric undernutrition, the great reduction in consumption of sweets and pastries, and a considerable increase in physical activity, especially among the females.

In France the general situation was similar to that in Belgium (Mahaux, 1946). Both the incidence and the severity of diabetes decreased in the first years of the war, and these changes were thought to be related to a reduction of obesity. The wide extent of the black market in France made for a large difference in diets according to economic circumstances. Among people who were economically well off, the diet was not severely restricted and the incidence and type of diabetes did not change. By 1943 the supply and distribution of food had improved and there was a general tendency to regain lost weight; at this time there was a deterioration in the diabetes picture. Mahaux (1946) emphasized the fact that all these changes did not involve the severe juvenile type of diabetes; the incidence of these cases was unchanged, and the patients did poorly on the restricted diets. At some times and places in France there were severe shortages of insulin; under such conditions those patients with the severe type of diabetes simply died forthwith.

In Germany, Oberdisse and Fleckenstein (1942) studied 151 diabetic patients in Würzburg in 1938 and in 1940. The estimated ration allowance for diabetics provided an average limit of 180 gm. of carbohydrate, 93 gm. of protein, and 100 gm. of fat, or a total of about 2000 Cal. daily. The patients studied by Oberdisse and Fleckenstein both in 1938 and in 1940 consumed considerably less than the carbohydrate allowance, but in practically all cases more carbohydrate was consumed in 1940 than in 1938. The clinical appraisal of these patients in 1940 indicated that 83 per cent of the cases with mild diabetes and 53 per cent of the cases with moderately severe diabetes were improved over their condition in 1938; none of the mild and only 4 per cent of the moderately severe cases of diabetes had deteriorated. In the small group of 13 severe cases of diabetes the gains were about equal to the losses. These data are summarized in Table 432.

Beckert (1940) showed that even in the first year of World War II there was a marked improvement in diabetes in Dresden. Promptly with the start of the war the rationing program, long prepared for the onset of war, went into effect, there was an immediate reduction in food, and the population generally lost weight. Beckert compared 604 diabetics in the summer of 1939 and the summer of 1940. A loss in weight was recorded in 62.2 per cent. Clinically, 45 per cent of the 604 patients were improved in 1940 and only 5 per cent were worse. The sugar tolerance was definitely improved in 25 per cent of the patients. During the winter of 1939-40, however, there was a transient deterioration in 30 per

TABLE 432

COMPARISON OF 151 DIABETIC PATIENTS IN WÜRZBURG, GERMANY, IN 1938 AND IN 1940. Blood and urine sugar values are given in mg. per 100 cc., insulin in the average number of units used daily, carbohydrate (CHO) as the estimated daily intake. (Oberdisse and Fleckenstein, 1942.)

Type of Diabetes	Blood Sugar (mg./100 cc.)	Urine Sugar (mg./100 cc.)	Insulin (units)	CHO (gm.)	Clinical Change in 1940
Mild (39 patients)					
1938.....	141	380		113	
1940.....	124	240		166	improved
Mild (8 patients)					
1938.....	168	440		107	
1940.....	159	860		112	unchanged
Moderate (48 patients)					
1938.....	160	240	38	107	
1940.....	126	530	31	135	improved
Moderate (39 patients)					
1938.....	168	490	30	108	
1940.....	161	790	39	133	unchanged
Moderate (4 patients)					
1938.....	159	290	24	116	
1940.....	188	1590	55	124	deteriorated
Severe (5 patients)					
1938.....	261	1810	65	112	
1940.....	186	1660	52	125	improved
Severe (4 patients)					
1938.....	190	1020	58	116	
1940.....	164	1590	81	140	unchanged
Severe (4 patients)					
1938.....	153	1140	40	122	
1940.....	165	1930	94	139	deteriorated

cent of the patients. Beckert considered the major change in the diet to be a reduction in fat and believed this to have been beneficial. It could be argued, of course, that the change in total calories was really responsible.

So far the evidence presented about diabetes in relation to nutrition has been limited to degrees of undernutrition which fall short of the severe condition of famine. In World War I those areas of Eastern Europe where there was real famine supplied no valid statistics pertaining to diabetes. On the whole, the same deficit of information characterizes World War II and its more widespread actual starvation.

Vitamins in Relation to Diabetes

Wherever there is mass caloric undernutrition there is the possibility that major changes in vitamin nutrition may occur. It appears that vitamin deficiencies in famine areas may be less frequent and less important than was feared at the start of World War II. The fact that when real food shortages occur there is usually recourse to the use of whole cereals and garden vegetables may mean an actual improvement in the vitamin quality of the diet. But in view of the substantial changes in diabetes in periods of food shortage, it is necessary to examine briefly the possible role of the vitamins.

In the past few years practically every ailment known to man has been claimed to be due to a vitamin deficiency or to be cured by vitamin administration. Diabetes mellitus has not been omitted. The known fact that certain vitamins of the B complex participate in fundamental reactions in intermediary carbohydrate metabolism suggests a point of attack. It has been reported that a rapid increase of carbohydrate intake and insulin dosage in diabetic patients may accentuate a slight pre-existing deficiency of B vitamins (Sydenstricker, Geeslin, and Weaver, 1939). This mildly interesting idea is a far cry from the claim of Biskind and Schreier (1945) that protracted deficiencies of the B vitamins play a primary role in the etiology of diabetes. The theoretical arguments advanced may be dismissed as largely irrelevant; the finding that diabetic patients who also have definite signs and symptoms of B complex deficiencies are improved by intensive vitamin therapy is hardly surprising. Guest (1946) pointed out that if Biskind and Schreier were correct we should find more diabetes in areas where B complex deficiencies are prominent, but that this is not the case. Lowry and Hegsted (1945) found no indication that the thiamine requirement is increased in rats made diabetic with alloxan.

It has been suggested that some of the complications of diabetes may be related to vitamin deficiencies. Root and Mascarenhas (1946), on the basis of entirely inadequate evidence from two cases of diabetic neuropathy, suggested that vitamin utilization may be inefficient in uncontrolled diabetes and so lead to a deficiency which aggravates or even provokes the neuropathy. Rudy and Epstein (1945) reported that diabetic neuropathy is frequently associated with signs of deficiency of the B vitamins and thought that the disordered metabolism of the diabetic may result in a vitamin deficiency without the diet being seriously at fault. On the other hand, from a careful review of the literature and a very large series of patients, Rundles (1945) concluded that B complex deficiency is not importantly concerned in the development of the neuropathic complication of diabetes. All authorities seem to be agreed that vitamins alone are of no value in treating diabetic neuropathy. In European countries where an amelioration of diabetes mortality resulted from food shortages, the incidence of complications such as neuropathy apparently decreased.

For many years it has been generally believed that yeast may be beneficial to diabetics. The insulin requirement of depancreatized dogs is increased on a diet definitely deficient in B vitamins, and this extra defect may be corrected either with yeast or with a mixture of B vitamins (Martin, 1937; Gaebler and Ciszewski, 1945). Dienst (1939), from trials with 6 diabetic patients without obvious signs of vitamin deficiency, concluded that the vitamin requirements of diabetics, particularly for thiamine, are higher than those of normal persons. It would be reasonable to expect that, in the past at least, many diabetic persons would have had low vitamin intakes because of the peculiar diets sometimes used for the control of their disease. Such dangers are now recognized by all competent physicians, and the dietary instructions to diabetics contain ample safeguards.

Consideration of the foregoing discussion and of the results of mass under-nutrition suggests that we must accept either one or both of two conclusions.

(1) European undernutrition of the two world wars did not involve deficiencies of those vitamins which may be related to diabetes; and/or (2) non-obese diabetics do not have more than ordinary vitamin requirements for the control of their diabetes.

The Nature of Diabetes

The mechanism and the significance of large reductions in diabetes mortality in times of food shortages and undernutrition can only be fully understood in the light of detailed knowledge of metabolism in diabetes and of the etiology of the disease. Until such complete knowledge is at hand we must be content with analyses which are somewhat speculative and largely descriptive. Many years ago the French began to talk about two major forms of diabetes — *diabètes maigres* and *diabètes gras*. Because the obvious biochemical faults are much the same in these two conditions and both have the same general response to insulin, there has been a strong tendency to consider them to be one and the same disease with the same etiology — to be distinguished possibly as developing in physiologically younger and older persons, respectively. This reasoning may open the way to major errors.

In any case it seems clear that caloric undernutrition is beneficial to the older and obese patients and is of less value or may be detrimental to the younger and non-obese patients. It is scarcely necessary to observe that, on the average, this distinction in patient types also corresponds to a difference in severity of the disease, or perhaps in the relative dangers of two related diseases. Lawrence (1946) noted that the usual obese diabetic is relatively resistant to insulin, does not readily develop ketonuria, and is improved by losing weight. He concluded that hyperglycemia results "from such an overfilling of the fat depots that they can no longer accept and absorb an excess of sugar from food." Just how this applies to the non-obese patient is not clear, but, descriptively at least, this might account for the diminished hyperglycemia and other improvements in times of food shortages.

National mortality statistics do not distinguish between obese and non-obese or between juvenile, adult, and senile types of diabetes. Regardless of the effect of undernutrition on the disease in young, non-obese patients, if undernutrition is advantageous to the older, relatively obese patient, the over-all figures would tend to show an improvement simply because there are so many more of the latter type.

All evidence now tends to show that a major or even the primary fault in diabetes is failure in fatty acid metabolism, particularly the synthesis of fatty acids from glucose. The same general conclusion emerges from studies on diabetes induced by pituitary extract (Young, 1945) and diabetes resulting from destruction of the islet cells in the pancreas by alloxan (Stetten and Boxer, 1944; Stetten and Klein, 1945). Hyperglycemia is simply an expression of the fact that there is an excess of sugar which cannot be stored or burnt at the moment. But the fatty acid catabolism which yields energy is not abnormal.

With considerable oversimplification, it is possible to describe as follows the case of the diabetic placed on an inadequate diet. He would have a lower blood

sugar concentration, and hence less glycosuria, simply because the excess of exogenous sugar is reduced. If he had large stores of fat in his body, his energy requirements might be met without difficulty and the general result would be improvement. In the absence of reasonable fat stores in the body, however, the results would be quite different, and there might be deterioration because of effective starvation and catabolic destruction of vital tissues. Such a picture may be satisfactory as far as it goes, but it does not make clear just why a maintained level of undernutrition should be particularly beneficial.

Cancer and Other Neoplasms

IN THE Middle Ages fasting and hunger cures were advocated in the treatment of tumors and "swellings," and other ailments. The view that dietary measures, generally involving some form of restriction, are efficacious in the control of cancer has never lacked proponents. A pretentious, rambling, and uncritical discussion is provided by Hoffman (1937). Neumann (1935) lists a large number of different dietary treatments for cancer, none of which, including his own, can command scientific support (cf. also Geréb, 1936). But in spite of the fact that the literature on the subject is overburdened with far more conclusions than facts, there is ample evidence that many types of tumor cells are more sensitive to undernutrition than the normal tissues of the host animal. The response is not dissimilar to that seen with radiation and some other noxious agents. The effect is not surprising in view of the familiar high metabolic demands of neoplastic tissues.

Incidence of Spontaneous Tumors in Animals

One of the first serious experimental approaches to the problem of the relationship between cancer and the caloric level was reported by Moreschi (1909), who found that a certain mouse sarcoma occurred less frequently in animals which were losing weight on a low caloric diet. The general fact that underfeeding results in fewer neoplasms in animals has been confirmed many times, and particularly striking reductions in tumor incidence have been obtained when mice from high cancer strains have been chronically underfed. Sivertsen and Hastings (1938), for example, found that only 16 per cent of chronically underfed mice developed breast cancers, while 88 per cent of their well-fed controls suffered from these tumors. McCay *et al.* (1939) observed fewer spontaneous cancers in underfed than in normal mice. Tannenbaum (1940, 1942a) obtained similar results in extensive experiments. Visscher *et al.* (1942) reduced the diet by one third and found no tumors up to 17 months of age in a mouse strain which on a normal diet showed a breast cancer incidence of 67 per cent. Similarly striking reductions in breast cancers were observed by White *et al.* (1944).

Wherever a reduction in cancer has been observed in such experiments, the reduction in dietary intake has been considerable. No effect on tumor incidence has been reported with only slight or moderate underfeeding, the general requirement being a chronic reduction in the diet by one third or more. Bischoff and Long (1938) observed no effect until the caloric intake was reduced to two thirds of the normal level, and they obtained really marked reduction in incidence only when the diet was reduced to half rations. Morris (1945a) pointed

out that definite effects have been obtained only when the diet reduction was severe enough to impair the growth of the animal's normal tissues.

It may be that the reduction in spontaneous breast cancers in underfed animals is, at least in part, a result of the suppression of ovarian function rather than a direct effect of undernutrition on the tumor-producing tissue (Morris, 1945b). These mammary tumors are beyond question greatly affected by ovarian hormones, and undernutrition has a profound effect on gonadal function. But other tumors which are not particularly influenced by these hormones are also affected by undernutrition, though to a smaller extent. Mice of a strain characterized by a high incidence of spontaneous leukemia showed a decreased incidence when they were underfed (Saxton, Boon, and Furth, 1944; Lawrason, 1944). Similarly, fewer spontaneous lung tumors appeared in underfed mice of a strain having a high incidence of such tumors, but the inhibition was only partial (Larsen and Heston, 1945).

It is questionable how far one may generalize from these results. The animals studied have been from strains having an extraordinarily high normal incidence of spontaneous tumors of a limited number of types. In any case it is clear that the simple undernutrition must be both severe and prolonged to be effective.

The Growth and Recurrences of Tumors in Animals

Moreschi (1909) observed that the sarcomas which did appear in his underfed mice developed much more slowly than normally. Similar results have been obtained by many other investigators. Rous (1914) suggested that a reduced rate of growth of the vascular bed would automatically retard tumor growth. Tannenbaum (1940) related the growth of tumors to physiological age. He calculated, without very good quantitative bases, that the rate of growth of breast cancers in underfed animals is not retarded if allowance is made for physiological age and time.

Recurrences after surgical removal of breast tumors are definitely fewer in mice that have been underfed for some time before surgery (Rous, 1915). Rous added the important observation that if underfeeding was started only after surgery there was no effect on the frequency of recurrences. Sugiura and Benedict (1926) surgically removed breast tumors from 28 mice maintained on normal rations and from 37 mice which received only two thirds as much food. Of the mice on full rations, 18 (64 per cent) had recurrences and 15 developed new tumors. In contrast, only 6 (16 per cent) of the underfed mice had recurrences and 6 developed new tumors.

The growth of nonspontaneous cancers likewise seems to be retarded or suppressed by underfeeding. Tannenbaum (1940, 1942a) and Lavik and Baumann (1943) presented evidence to show that it is more difficult to induce cancers by carcinogenic agents in underfed animals and that if cancers do appear in such animals the tumors grow very slowly. Much the same is true regarding transplants. Sugiura and Benedict (1926) found that transplants of the Flexner-Jobling strain did not survive as frequently and grew more slowly in mice fed one third less than the normal caloric intake.

In general, it appears that the growth of many neoplasms is depressed by

severe underfeeding. Even mice with certain leukemias live longer when underfed than they would otherwise (Flory *et al.*, 1943).

Calories versus Other Dietary Factors

The cancer literature pro and con for almost every conceivable dietary factor is enormous, but the quality of the studies is almost inversely proportional to the mass of the printed matter. The potential value of many of the studies is much reduced because of lack of control of caloric intake (e.g. Waterman, 1938). The only aspect of the question which is of concern here is whether the quality of the diet exerts any important influence on the effect of caloric undernutrition. In the absence of relevant and acceptable information on man, reliance must be placed on the animal studies, which, fortunately, seem to be substantially consistent.

Bischoff, Long, and Maxwell (1935) and Bischoff and Long (1938) were unable to influence the rate of growth of sarcoma 180 by varying the proportions of carbohydrate, fat, and protein as long as the caloric intake was kept constant. Tumors retarded by underfeeding resumed the normal rate of growth when the diet was increased to normal by the addition of either fat or carbohydrate. Tannenbaum (1940) found that vitamins added to a calorically deficient diet had no effect on the frequency or growth of tumors in animals. Visscher *et al.* (1942) used diets ample in all known dietary factors and equal to the control diets except for caloric content. The reported increase in tumors on high fat diets (e.g. Waterman, 1938; Tannenbaum, 1942b) does not seem to be particularly related to the caloric intake. Incidentally, starving animals are subsisting on a relatively high proportion of fat (endogenous), so the mere metabolism of fat per se does not seem to favor cancer development.

Evidence in Man of an Effect of Calories on Cancer

Many investigators have been convinced that relative obesity favors the development of cancer (cf. Hoffman, 1937). Actuarial records in the United States indicate that persons who were relatively overweight at the time of taking out life insurance have a higher mortality from cancer than do persons who were not obese when they became insured. Dublin (1929, 1932) analyzed the records for 192,000 men who were 45 years of age and older. In comparison with men of average weight, he found a 9 per cent excess in cancer mortality for men 5 to 14 per cent overweight, a 24 per cent excess for men 15 to 24 per cent overweight, and a 29 per cent excess for men 25 per cent or more overweight. The men who were 5 to 14 per cent underweight had about 3 per cent greater mortality from cancer than the men of average weight, but for those who were as much as 15 to 50 per cent underweight the mortality was about 15 per cent less than in the average weight group. Later analyses provided general confirmation of these findings and extended them to include women (Dublin and Marks, 1938; Hunter, 1939).

If these actuarial findings can be taken to indicate a causal relationship, anything that would tend to reduce actual obesity might be expected also to result in a lowered average mortality from cancer. But merely a small reduction

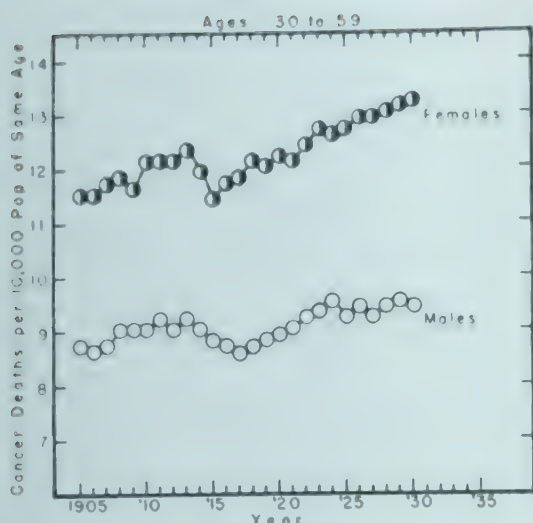


FIGURE 142. MORTALITY RATE FOR CANCER IN GERMANY, from 1905 through 1930, for persons aged 30 through 59, inclusive (Stupening, 1937).

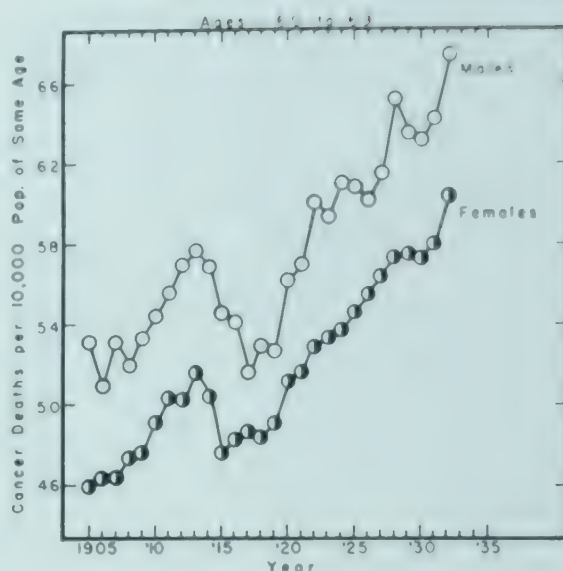


FIGURE 143. MORTALITY RATE FOR CANCER IN GERMANY, from 1905 through 1930, for persons aged 60 through 69, inclusive (Stupening, 1937).

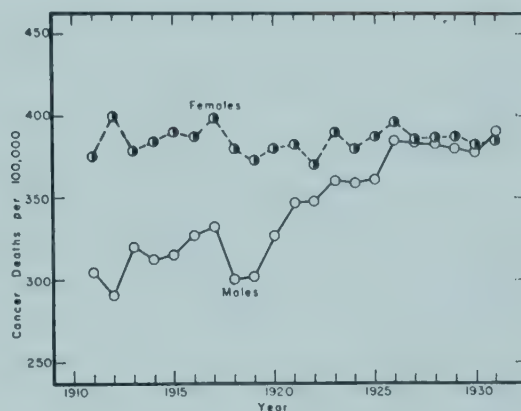
below average weight might have little or no effect. In any case, cancer mortality data for periods of food shortage should be illuminating.

Unfortunately, cancer statistics in famine areas are often not very good, nor has this point received adequate analysis. In 1919 it was suggested that cancer mortality in Germany did not seem to be affected by the undernutrition of World War I (Rubner, 1918), but such impressions are obviously valueless. The actual course of cancer mortality in Germany from 1905 to 1932 was summarized by Stupening (1937), but he did not discuss the question of diet. The data are depicted in Figures 142 and 143.

From 1905 to 1914 cancer mortality in Germany was gradually rising for both males and females between the ages of 30 and 60 (Figure 142). It was rising more rapidly for the age group between 60 and 70, the general level for both sexes in this group being about 10 to 12 per cent higher in 1913 than in 1905 (Figure 143). In 1914, however, there was a slight decline in cancer mortality for both sexes and for both age groups. Since there was no real food stringency in Germany in 1914, it is scarcely possible to ascribe this small change to undernutrition. But in the years 1915–18 the cancer mortality rate was definitely low in all groups, and thereafter the previous upward trend was resumed. The data certainly suggest that cancer mortality was favorably influenced by the war, and the possibility that this was related to general undernutrition is obvious.

In Austria the data are rather similar to those for Germany. In the town of Graz the cancer mortality rate showed irregular fluctuations for both males and females until 1917, but in 1918 it fell to a record low for the 25-year period, 1910–34. It tended to remain low until 1924, after which it showed a definite rising trend (Burkard, 1940). It is interesting to note that the food shortage developed later and lasted longer in Graz and other parts of Austria than in Germany. Burkard pointed out that the population of Graz and its environs re-

FIGURE 144. CANCER MORTALITY RATE OF THE CORRESPONDING AGE AND SEX, per 100,000, for men and women from 45 to 74 years of age insured in the Industrial Department of the Metropolitan Life Insurance Company (U.S.A.) (Dublin, 1932).



maintained numerically constant over the period from 1910 to 1934 but that the age composition of the population steadily changed; by 1934 persons over 50 years of age comprised 32.8 per cent of the population as compared with 21.4 per cent in 1910. This difference should result in a tendency toward increased cancer mortality and might account for the upward trend after 1923; obviously it could not explain the drop toward the end of the war and immediately thereafter.

Russell (1931) analyzed the cancer mortality for persons over 35 years of age in Britain for the period 1891 to 1927, but his use of a 3-year period to represent each decade obscured the true effect, if any, of World War I. However, the data do show a real decline in the cancer mortality rate for the period 1920–22 as compared with the trend line established for the decades represented by 1870–72, 1880–82, and subsequent 3-year units.

In the United States the experience of the Industrial Department of the Metropolitan Life Insurance Company from 1911 to 1931 for men and women aged 45 to 74 years is summarized in Figure 144 (data from Dublin, 1932). Over these 21 years there was no important trend in the female mortality rate, but the rate among males is interesting. A general trend upward characterized male cancer mortality for the period as a whole, but this was sharply interrupted by a decline in 1918 and 1919. Dublin (1932) was inclined to relate this to the fact that all non-influenza deaths tended to diminish during the influenza epidemic in the latter half of 1918 and early 1919. But why should this effect be so clear among males and not among females? Besides the influenza epidemic, this period, as well as the year 1917, was characterized by greater intensity of physical work, a large increase in the number of insured persons, and little or no caloric shortage. Perhaps there was a reduction in the prevalence of obesity, but there are no adequate data on this point. It may well be that the change in cancer mortality reflected a change in the character of the insured population. In any case these data illustrate the difficulty in the use of insurance statistics and suggest the need for caution in the analysis of mortality data in general.

We have few useful statistics from World War II, but the records from Greece are of much interest. In the metropolitan population of just under one million persons in Athens and Piraeus, cancer mortality steadily declined after the development of famine conditions. The data given by Valaoras (1946) are summarized in Table 429. The total cancer mortality was 990 in 1940, 974 in

1941 (the first year of famine), 899 in 1942 (estimated from 72 per cent of the population and corrected to 100 per cent), and 820 in 1943. In contrast, deaths from all other major causes rose very sharply in 1941 and 1942.

The Trend of Present Evidence

Cancer mortality statistics are imperfect at best, and the facts required for their proper analysis are seldom available in full. But it is of interest that the data which are at hand tend consistently in the same direction — a direction that is consistent also with the results of animal experiments. The present evidence is at least strongly suggestive. Whatever may be the reason or mechanism involved, prolonged severe undernutrition in both man and animals seems to depress cancer incidence, or growth, or both. It is not unreasonable to suggest that dietary restrictions, perhaps coupled with increased physical activity, may be beneficial in cancer control (cf. Potter, 1945). But the severity of the dietary restriction required to produce positive effects in animals is such that estrus is inhibited and normal growth markedly retarded (Morris, 1945a). The practical difficulties in human application are obvious (Rusch and Baumann, 1946). In instances where cancer mortality statistics indicate a real effect in times of food shortage, food restriction has gone beyond the point of merely preventing the development of obesity.

It is pertinent to note that in many types of cancer general undernutrition of the host is usually produced as a tumor develops; the improvement of the nutritional state in these patients is generally accepted to be a major requirement in their medical care. The effect of the nutritional state of the patient on the growth of tumor is itself seldom considered.

Diets for Rehabilitation, with Special Reference to the Minnesota Experiment

THE problem of the nutritional rehabilitation of an undernourished person or a semi-starved population demands consideration of the character and amount of foods to be fed. Upon the nutritional characteristics of the rehabilitation diet will depend the rate, extent, and economic cost of recovery. In the vast majority of cases due regard must be given also to practical considerations—the local food supply, cost and availability of supplementary foods, established eating habits, and the methods for distribution. The exact state of nutriture at the outset is a major determinant. The starting point is fixed by the extent, qualitative character, and duration of the undernourished state. Questions of age, sex, and concurrent disease must enter the calculation. From the strictly nutritional standpoint, the major questions in selecting the rehabilitation diet for famine relief have to do with calories, proteins, and vitamins. Generally speaking, minerals do not seem likely to be limiting factors in most famine areas. For rehabilitation feeding, fats are relatively unimportant, except as a source of energy and for purposes of morale. It should be noted that calories derived from fat are expensive. In starvation areas fats are likely to be available only in limited quantities for a considerable length of time.

Most of the information on which this chapter is based comes from the Minnesota Experiment and can be found in the several chapters dealing with the separate functions and measurements. It seems wise, however, to provide here a synopsis of the more important results which are pertinent to the evaluation of the rehabilitation diets so that these facts may be considered as a unit, unencumbered by the details which are necessary for the complete presentation of the effects of starvation and rehabilitation on a given function. Furthermore, special problems in the interpretation of changes in specific functions and their relationships to rehabilitation present themselves. For example, what changes in what functions can be considered “good”?

The individuals who bear the heavy responsibility of administering a relief program in the field may ask for specific recommendations. They will not find a ready-made solution to their problems in this chapter. We had hoped that useful field studies on rehabilitation diets would result from relief work after World War II and that these could be integrated with the experimental data. In spite of considerable effort to find such materials, extremely little useful information has come to our attention.

The evaluation of rehabilitation diets, and of the role of the quantity and character of the individual nutrients, must be made from their effects on undernourished people. There is no reason to suppose that the demands or wishes of the undernourished persons themselves will offer a good guide as to what they should have; as a matter of fact, in both primary and secondary undernutrition, catering to such wishes may be dangerous as well as inefficient. It is necessary, then, to base judgment on the recorded changes in the characteristics of individuals which have resulted from the provision of specified rehabilitation diets.

Selection of Variables for Evaluation of Rehabilitation Diets

Any function whose measurement is to be used to evaluate the effectiveness of different rehabilitation diets must satisfy several criteria. It should be a function which has been well defined for the pre-starvation state; it should have been markedly influenced by semi-starvation and should respond to dietary rehabilitation; and, finally, it should be susceptible to clear-cut interpretation. The latter criterion raises surprisingly thorny problems. For the present consideration of the Minnesota Experiment, we have selected representative functions from those which seem to meet these criteria most completely.

Stable reference points for characterizing the control values were ensured by taking the mean of 2 observations on each variable during the last 2 weeks of the control period. All these functions changed dramatically during the 6 months of semi-starvation. The relative amount of change among the several variables is shown in Table 433, where the starvation increment is evaluated in the light of the distribution found in the group in the control period. It will be noted that the basal metabolic rate, hemoglobin, endurance time, Depression score, and body weight, in the order named, showed the largest changes due to starvation as judged by the displacement of the mean, with the standard deviation of the scores during the control period serving as a unit of measurement. All the variables presented showed statistically highly significant changes during the 12 weeks of rehabilitation.

TABLE 433

MAGNITUDE OF CHANGES IN SEMI-STARVATION. C = mean at control; SD_C = standard deviation of the control values; dS24 = mean change after 24 weeks of starvation. Statistically, the mean difference was highly significant for all functions included in this table.
(Minnesota Experiment.)

	C	SD_C	dS24	$\frac{dS24}{SD_C}$
Body weight (kg.)	69.39	5.85	16.82	2.88
Body fat (kg.)	9.84	(4.18)*	6.80	(1.63)
Fat-free body weight (kg.) ..	59.55	4.22	10.02	2.37
Hemoglobin (gm./100 cc.) ...	15.12	0.88	3.43	3.90
Basal metabolism (cc. O_2 /min.)	288.09	13.50	88.94	6.59
Basal pulse rate (beats/min.) .	55.19	6.49	17.88	2.76
Handgrip (kg.)	58.16	7.59	16.38	2.16
Endurance time of run (secs.) .	241.5	52.9	191.3	3.62
Depression (MMPI score)	54.25	6.48	19.62	3.03

* Distribution not normal; skewed to the right.

Rehabilitation Diets—Minnesota Experiment

Before presenting data illustrating the effects of the rehabilitation diets, it seems useful to review briefly the nutritional design of this period in the Minnesota Experiment. The caloric intakes in the different groups are given in Table 434. It will be recalled that the factorial design allowed the study, over a period of 12 weeks, of the effects of caloric intake at 4 levels for 8 men each, the effects of a protein supplement on 2 groups of 16 men each, and the effects of a vitamin supplement on 2 groups of 16 men each. Details of the design and of the diet were presented in Chapter 4. The average daily supplement of protein (part casein and part soybean protein) was 20 gm. per man. The vitamin supplement consisted of one Hexavitamin pill a day, which provided, in addition to the diet itself, from one half to one full daily allowance, as recommended by the National Research Council (1943), of the following vitamins: A and D, thiamine, riboflavin, niacin amide, and ascorbic acid.

Results—Minnesota Experiment

Table 434 presents the changes observed in the several functions for each diet group during 12 weeks of rehabilitation, expressed as percentages of the changes during starvation. The significance of the differences between the various diets is presented in Table 435.

The differences between the *absolute* rehabilitation (R12) and starvation (S24) values, not the percentages of recovery, were used in evaluating the statistical significance of the differences in recovery between dietary groups. Marked differences between the rehabilitation dietary groups in the amounts of starvation deterioration were the exception. The mean Depression scores are among the most striking in this respect, with mean semi-starvation increments of 22.75 in the Z group and 16.75 in the T group; 17.06 in the U group and 22.19 in the Y group. Handling such cases as "matched" groups is obviously a questionable procedure, and the statistical treatment of recovery values would gain by the refinements of covariance analysis. In the majority of functions the ordinary analysis of variance of absolute rehabilitation changes was a satisfactory technique of statistical evaluation.

In the preceding chapters the significance of recovery differences between caloric groups (Z, L, G, T) was evaluated in terms of paired comparisons. With 4 groups, 7 comparisons are possible. For summary purposes, in Table 435 an over-all F-test was computed which tests the significance of the differences between the recovery means of all 4 caloric groups considered simultaneously. The between-group variance is associated with 3 degrees of freedom. In the case of body weight, endurance, basal metabolic rate, and body fat, the effect of calories is clearly demonstrated.

It is pointed out in the Appendix section on statistical methods that the F-tests used above take into account the *magnitude* of the mean differences between dietary groups but not their *direction* (in the case of 2 groups) and/or *order* (in the case of 3 or more groups). Consequently, it appeared useful to determine the regression line fitted to the recovery means of the 4 caloric groups and to test the significance of the departure of the slope from the horizontal

TABLE 434

RELATIVE MAGNITUDE OF RECOVERY AFTER 12 WEEKS OF DIETARY REHABILITATION. The recovery values are expressed as percentages of semi-starvation changes. (Minnesota Experiment.)

	Dietary Groupings								All 32 Subjects
	Calories (8 subjects each)			Proteins (16 subjects each)			Vitamins (16 subjects each)		
	Z (+0)	L (+400)	G (+800)	T (+1200)	U (basal)	Y (extra)	P (basal)	H (extra)	
Average daily caloric intake									
In semi-starvation	1543.6	1571.0	1619.1	1544.3	1582.6	1556.4	1612.1	1526.9	1569.5
In rehabilitation	2377.9	2692.4	3122.6	3391.9	2844.2	2948.2	2915.0	2877.5	2896.2
Body weight	21.0	29.6	40.7	57.2	35.9	37.8	40.0	33.9	36.8
Body fat	17.1	42.5	45.9	94.8	49.9	48.5	55.0	45.0	49.3
Fat-free body weight	27.3	22.3	36.4	32.0	25.0	31.5	31.4	25.1	28.3
Hemoglobin	32.6	29.9	35.7	26.5	28.4	34.0	36.8	25.2	31.1
Basal metabolism	37.2	45.8	55.1	65.1	52.1	46.3	51.5	46.9	49.2
Basal pulse rate	60.0	71.4	67.4	75.6	68.3	70.0	62.4	76.7	69.0
Handgrip	15.7	24.3	47.0	47.4	34.8	30.6	25.4	39.9	32.8
Endurance	18.0	32.6	43.4	45.7	34.7	34.0	35.2	33.5	34.3
Depression	9.9	44.0	51.0	72.4	26.4	53.8	43.8	40.1	41.9

TABLE 435

EVALUATION OF THE STATISTICAL SIGNIFICANCE OF THE MEAN DIFFERENCES BETWEEN DIETARY GROUPS, in absolute scores, not percentages. The F values represent the ratios of the variances (mean squares), V, due to dietary factors, and the replicate (error) variances, V_{rep}, (Minnesota Experiment.)

	Dietary Factors				V _{rep}
	Calories (Z, L, G, T)	Calories (linear slope)	Proteins (U vs. Y)	Vitamins (P vs. H)	
Body weight					
V	44.14	132.13	0.04	1.44	3.02
F	14.62[**]	43.75[**]			
Body fat					
V	31.59	92.72	5.78	0.72	2.28
F	13.86[**]	40.67[**]			
Fat-free body weight					
V	2.43	3.48	4.80	4.20	2.51
F					
Hemoglobin					
V	0.10	0.02	0.09	1.40	1.10
F					
Basal metabolism					
V	1457.18	4253.91	205.03	234.53	160.59
F	9.07[**]	26.49[**]			
Basal pulse rate					
V	42.70	82.66	30.03	5.28	29.47
F					
Handgrip					
V	42.92	99.22	15.12	55.12	28.50
F	1.51	3.48			
Endurance					
V	3563.34	7507.6	84.5	105.1	626.1
F	5.69[**]	12.00[**]			
Depression					
V	142.70	375.16	442.53	0.28	87.66
F	1.63	4.28	(5.05[*])		

* One asterisk in brackets [*] attached to an F value indicates statistical significance at the 5 per cent level; [**] indicates significance at the 1 per cent level.

level. The resulting between-group variance is associated with 1 degree of freedom. The F-tests are significant for those functions, and only those functions, in which caloric groups were differentiated by the simpler analysis of variance. In the case of the Depression scores the F value approximates but does not reach the 5 per cent level of significance.

The comparison of basal protein and extra protein groups and of basal vitamin and extra vitamin groups is statistically a straightforward matter. The between-group variance is associated in both cases with 1 degree of freedom. The F-tests of statistical significance of the differences are obtained as the ratio of the between-group variance to the replicate (or error) variance, V_{rep}, associated with 16 degrees of freedom. As shown in Table 435, the protein supplement indicated a significant difference (at the 5 per cent level) only in the Depression score. This difference may be regarded, in part, as an artifact due to the unequal group responses to starvation. In view of the lack of supporting evidence from

other items, we are forced to conclude that within the range of protein supplementation used in the Minnesota Experiment the protein supplement had no real beneficial influence on the course of rehabilitation.

The vitamin supplement had no statistically significant beneficial effect on any of the functions studied. It should be remembered that the experimental design included many items in the biochemical, physiological, and psychological areas which have been demonstrated to be sensitive to vitamin deficiencies, particularly of thiamine (Keys *et al.*, 1945). The endurance time and the Depression score have been included in Tables 434 and 435. Both these items showed enough change in semi-starvation to have been able to reflect any important benefits derived from the vitamin supplementation. The unequal group responses to starvation which complicated the analysis of the protein effect on the Depression score were not present in the 2 vitamin groups.

Comment on Rehabilitation Diets—Minnesota Experiment

In view of the large number of items studied in the Minnesota Experiment, it may be surprising that greater differentiations were not obtained among the 4 calorie levels and that no differentiation appeared between the 2 levels of proteins and the 2 levels of vitamins. It might appear that, with the exception of the caloric effects on several items, the basic rehabilitation diet (Z, U, P, with respect to calories, proteins, and vitamins) was sufficient to produce near-maximal rates of recovery. On the other hand, the limitations in the present methodology of functional measurement must be admitted. Some of the methods, though applied here with the utmost rigor, are relatively crude and unreliable. More important, perhaps, is the probability that consideration of single functions one by one may be misleading. The most advantageous value for any one variable may be largely fixed by the other current conditions existing in man. We have remarked that both basal metabolism and heart rate must return—that is, increase—to the pre-starvation level before we will admit that recovery has been achieved. But a metabolic rate high enough to overload the heart by its demand for circulation is obviously bad. Conversely, a heart rate more rapid than is required by the metabolic demand is undesirable. Presumably, some index involving both B.M.R. and heart rate would be a more just measure of recovery than either taken alone. We have not attempted such analyses here; the possibilities seem large, but so are the difficulties of both mathematical and physiological analysis.

Other examples of the difficulty of interpreting the biological desirability of changes in rehabilitation may be cited. There seems to be no question of the desirability of obtaining relief from mental depression, or of increasing strength or endurance. An increase in the body weight of the starved is also desirable, but even here one may suggest that increases in weight due to accumulations of fat and water do not represent proper rehabilitation. The interpretation of hemoglobin concentrations is also not as clear as one would like. Keys *et al.* (1938) have observed that individuals with hemoglobin concentrations in the lower limits of normal make a better adaptation to the stress of high altitude than those in the upper limits of the normal range. Moreover, Adcock *et al.* (1948) have

shown that physically active—and presumably more “fit”—men have lower hemoglobin concentrations than comparable sedentary men. In other words, there are reasons for believing that low normal hemoglobin values are “better” than high normal values.

In those items which underwent marked changes in starvation but did not distinguish between rehabilitation diets during recovery, it is occasionally possible to make an intelligent guess as to why this failure occurred. This is the case in the hemoglobin concentrations, which showed one of the largest declines during starvation but did not differentiate between either caloric or protein groups. The rate of hemoglobin synthesis is notoriously slow even under ideal conditions (Whipple, 1942), and it appears likely that the rate of synthesis and not the supply of nutrients was the limiting factor in this case.

There are suggestions in the data that high caloric intakes are not well utilized. The body weight and the fat content distinguished sharply between the caloric groups while, on the other hand, the fat-free body weight failed to demonstrate any definite and progressive advantage for calories. Examination of the actual data reveals that the top group gained a disproportionate amount of fat. The same group made a relatively small gain in endurance. The failure of strength measurements to distinguish between the caloric levels suggests that the recovery of muscle tissue was not influenced by calories in the same manner as body weight. Finally, it should be remembered that evidence has been presented in Chapter 28 that excessive caloric intakes may impair cardiac function.

The finding that a protein supplement of 20 gm. a day provides no advantage in rehabilitation is in no way a denial of the general principle that the isocaloric substitution of large quantities of protein is useful in the presence of caloric restriction both during growth and in adults (Bosshardt *et al.*, 1948; Benditt *et al.*, 1948; Elman *et al.*, 1945). The protein supplement provided in the Minnesota Experiment was only 25 per cent of a protein intake which was already at a moderately good level, with 75 (Z group) to 100 (T group) gm. of protein a day. Compare this with the situation studied by Bosshardt *et al.* (1948), who used a relatively low protein intake in growing rats and mice in the presence of caloric restriction and compared the protein deposition in similar animals whose protein intake was tripled on an isocaloric basis. Those who feel that the high protein content of the basal rehabilitation diet represents poor experimental design should remember that in areas where grain cereals and potatoes are readily available for rehabilitation feeding, a protein content of the diet similar to that used in the Minnesota Experiment is unavoidable. It will be noted that the effects of protein were studied at an average daily caloric intake of 2900. This can be considered a “caloric restriction” in the rehabilitation period, as demonstrated by the fact that men receiving 3100 and 3400 Cal. a day did better than those who had smaller quantities. The results reported here do not rule out the possibility that small protein supplements would have had a measurable effect if tested at a lower caloric level. It must be expected, however, that such an effect, if it had existed, would have been small. In the practical situation, it would seem that the relatively high cost of providing special protein feedings as compared with the cost of supplying additional calories far outweighs any possible ad-

vantage which might be gained by small protein supplements at lower caloric levels.

Some comment should be made on the role of protein in the caloric effect demonstrated in the Minnesota Experiment. It was not possible to maintain isoprotein intakes in the several caloric groups. The T group received 25 gm. of protein a day more than the Z group. We have demonstrated that a difference in protein intake of this order of magnitude had no effect on rehabilitation. It seems likely that the protein-sparing effect of calories was not large, since the caloric intake of the upper groups appeared adequate. However, it is at least possible that this increase in protein intake, coupled with the protein-sparing action of additional calories, may have made a small contribution to the effect of calories. In spite of this, we do not wish to qualify our conclusion that the caloric intake is the single nutritional element of highest importance in rehabilitation feeding of persons starved on a European type of famine diet. While theoretical nutritionists may object to this conclusion, it is correct on the practical level since any increase in cheap calories (i.e., grains, cereals, and potatoes, but not refined sugar) must of necessity be accompanied by an increase in the protein (and the vitamin) content of the diet.

We wish to point out that in any practical applications of the conclusions derived from the Minnesota Experiment, it should be kept in mind that these results were obtained for groups of young white men who had lost one quarter of their body weight in a 6-months' period on a Northern European type of famine diet. Differences in the rate of weight loss, the extent of weight loss, the type of diet eaten during the starvation period, the dietary history before starvation began, and the genetic background of the starved individuals might lead to different dietary requirements for optimal rehabilitation.

Problems of Early Refeeding—Field Observations

We have pointed out that no special difficulties arose in the early period of refeeding in the Minnesota Experiment (see p. 599). With similar degrees of starvation in young men a simple increase in ordinary foodstuffs should be satisfactory for relief feeding but some caution should be exercised to ensure that the early rehabilitation diet is not excessive in bulk or calories in comparison to the diet preceding the institution of relief. In a more desperate state of inanition, however, it might be thought that very different problems would arise and that special diets and devices for realimentation would be necessary.

In the dark days of early 1945 when planning was in progress for the relief of the western Netherlands and the German concentration camps it was believed that many of the famine victims would be unable to tolerate ordinary foods or even to digest any nutrients given by mouth. The viewpoints and subsequent developments have been reviewed by Burger, Drummond, and Sandstead (1948). On the urging of the experts attached to the Supreme Headquarters of the Allied Expeditionary Force (S.H.A.E.F.), the Nutritional Advisory Committee, and the Dutch Advisory Committee, strenuous efforts were made to prepare materials and apparatus for intravenous and oral tube feeding. Vigorous action was taken in March 1945 by the Directorate of Medical Supplies (Minis-

try of Supply) in the United Kingdom and various manufacturing firms gave enthusiastic cooperation. Large amounts of 5 per cent protein hydrolysate solution, 10 per cent glucose, and other materials were assembled.

The official Dutch report (Burger, Drummond, and Sandstead, eds., 1948, p. 56) concluded that the eventual operation of the emergency feeding teams was seriously affected by two false assumptions: "That patients in severe starvation states cannot swallow and digest simple foods, and that fragmentary intelligence, mostly from non-medical sources, is of real value in assessing a medical situation." Actually, it was found that the great majority of starving persons, both children and adults, could immediately take normal food. In most cases the appetite was good and there were no serious digestive difficulties if the quantity of food eaten was kept within moderate bounds.

In the Netherlands and with the victims of starvation in the concentration camps, intravenous feeding was used enough to provide a reasonable measure of its value. In some cases it seemed to be beneficial but in others the experience was definitely unfavorable. The same was true of the experience with "predigested" foods and protein hydrolysates administered orally. Comparison of the results of these methods of refeeding with those obtained with simpler oral foods, particularly skim milk powder and water, indicated no advantage — and some real disadvantages — in the more elaborate arrangements. "There is ample evidence in the investigations recorded here that the gastrointestinal tract of such persons can digest separated milk and even a large quantity of fats (butter) with a facility that was not expected. Only in the very last stage of starvation, when the patient was almost moribund, was direct feeding of no value; indeed, experience in the western Netherlands indicated that there is no treatment available that will resuscitate such cases" (Burger, Drummond, and Sandstead, eds., 1948, p. 165).

In a sense, the experience with the use of elaborate procedures and highly specialized materials for early refeeding is disappointing. Intravenous and tube feeding, predigested (and expensive) concoctions, seem to be seldom, if ever, required in simple starvation. Actually, the situation should be a cause for rejoicing. In the great majority of starving patients simple foods, taken by mouth in small quantities at frequent intervals, are more than merely well tolerated; they are usually efficiently utilized and rapidly start the patient on the road to nutritional recovery. Thereafter the nutritional rules that apply to growing children may well be followed for the long period required to achieve the full degree of rehabilitation.

Appendixes

NOTES ON THE APPENDIXES

Appendix I. METHODS

Appendix II. DETAILED DATA FROM THE MINNESOTA EXPERIMENT

Appendix III. WARTIME DIETS AND RATIONS

Appendix IV. SOME NOTABLE FAMINES IN HISTORY

*"Gaunt with hunger and with want they gnaw herbs in the wold.
. . . They wander like the wild ass in the desert, roaming in
search of food – for the children have no bread – till vigor fails
and their total strength is gone."*

JOB.

*"Non rationem patitur nec aequitate mitigatur nec ulla prece
flectitur populus esuriens" (Hungry people will not endure rea-
son, they will not listen to justice, nor will they bend to any
prayer for mercy).*

SENECA.

*"'Only the truth of number is eternal,' said the black girl. 'Every
other truth passes away or becomes error, like the fancies of our
childhood, but one and one are two and one and ten eleven and
always will be. Therefore I feel that there is something godlike
about numbers.'"*

GEORGE BERNARD SHAW, in *The Black Girl
in Search of God.*

*"Natural Science is a branch of knowledge by general consent
not primarily based on the a priori. It derives essentially from
details. It amasses them and lives on and by them."*

CHARLES SHERRINGTON, in *Man on His Nature*
(1941), Chapter 1.

Notes on the Appendixes

THE methods used in the Minnesota Experiment were indicated, where possible, in the text proper or identified by reference to the literature. In some instances more complete description and explanation appeared essential. These are presented in the first section of the Appendix.

The Minnesota Experiment photographs include examples of full-body, three-view pictures that were used in the photographic analysis of the body build and enlarged views of the faces; they indicate the general appearance of the subjects.

The data from the Minnesota Experiment are unique, not only in regard to specific information on starvation, but also in the wider area of human biology which has to do with the characteristics of individuals and the interrelationships between these characteristics. The analyses of these data for the purposes of these volumes of text by no means exhausted the value of the material for other analyses which may be made for other purposes and from other points of view. Accordingly, the individual data are presented in a series of tables from which an unusually complete picture of individuals may be gained.

Details of diet in studies on man are always troublesome to report accurately and completely, but some readers will want these for full understanding or for personal use. In spite of much effort, the dietary data from the Minnesota Experiment are not complete in all respects regarding specific nutrients which may, in the future, turn out to be of some consequence.

The Appendix on Wartime Diets and Rations is only a sampling of diets which have produced various degrees of undernutrition in recent times. It is surprising to discover, on critical examination of the literature, how little in the way of accurate dietary information there is available for areas and periods of food shortage. As far as possible we have selected for tabulation here only those dietary records which cover the whole subsistence of the people involved. To these have been added the composition of Red Cross and CARE parcels used to supplement the diets in some prison camps.

The list of Notable Famines in History is a sampling of the world's experience up to the 20th century.

APPENDIX I

Methods

Clinical Examination

THE continuous program of special tests and measurements served as a running record of the status of the subjects in the Minnesota Experiment. Besides the laboratory procedures, this program provided for recording (and discussing) subjective complaints. In addition, at frequent intervals throughout the Minnesota Experiment each subject was seen briefly by a resident physician who noted the general appearance and posture, examined the heart and lungs with the stethoscope, checked the mouth, tongue, throat, and ears, examined the skin, and tested the feet and ankles for edema. On these occasions the examiner also reviewed the X-ray films of the chest which were taken at intervals of a month to 6 weeks. At the end of semi-starvation (S24) and again after 6 weeks on the rehabilitation regimen (R6) more detailed examinations were made in which each man was seen by a team of 3 to 6 examiners.

In these special examinations particular attention was paid to the skin, eyes, mouth, and gums. Tendon reflexes (patellar and Achilles) were tested. The character of locomotion and the performance of rising from a squat and of stepping up onto a chair were noted. Cutaneous kinesthesia was tested by pinpricks and by stroking with a wisp of cotton. Gingival sensitivity was tested by pressure with a flat-tipped stylus. Vibratory sense was estimated by a tuning fork. Deep muscle sense was checked by firm digital pressure in grasping the gastrocnemius-soleus group of tissues in the lower leg and the quadriceps in the upper leg. Tension, texture, color, warmth, moisture, and irregularities of the skin on all parts of the body were examined. Lips, mouth, gums, tongue, and the oral tissues were carefully inspected. Eyes were examined with regard to reaction to light and movements of the eyeball, and the appearance of the cornea and conjunctiva was examined with a hand lens and a projected beam of light. The appearance and feel of the hair in the fingers were noted. Each man was also questioned carefully about peculiarities of sensation, bowel movements, sleep, and general sense of well-being.

Physical Anthropology

MEASUREMENTS ON THE LIVING

The dimensions measured and the procedures used were as follows:

(1) *Standing height* (or *stature*). The subjects stood on a horizontal, firm floor, maintaining an erect posture. The feet were parallel. The heels, buttocks, and shoulders were in contact with the wall. The head was free, with the axis of

vision horizontal. The upper limbs were pendant, lightly touching the sides of the thigh, with the palms of the hands turned inward and the fingers pointing downward. The standing height is the distance from the vertex, the highest point at the top of the head in the mid-sagittal plane, to the floor. It is well known that stature exhibits diurnal variations, decreasing during the wakeful hours (Backman, 1924). For this reason all anthropometric measurements were done during the morning hours.

(2) *Sitting height.* The subjects were seated on a horizontal, firm stool. The knees were flexed. The trunk was in contact with the wall at both the scapular and the sacral region. The position of the head was the same as for standing height (item 1). The sitting height is the distance from the vertex to the level of the top surface of the stool.

(3) *Bi-deltoid diameter.* The bi-deltoid diameter was defined as the maximum distance between the right and the left prominence formed by the deltoid muscles. The calipers just touched the skin surface. The measurement has a bony and a muscular component and so is not well suited for most anthropometric work. In the present experiment it was a sensitive index of the changes in muscle mass and in subcutaneous fat.

(4) *Bi-acromial diameter.* The bi-acromial diameter is a measurement of the breadth of the shoulder girdle. It was obtained as the distance between the lateral borders of the acromial process of the two scapulae. The subject stood erect but with shoulders relaxed; this is an important condition for making the measurement since increased tension of the muscles involved in upward and forward movement of the shoulders tends to decrease the bi-acromial diameter.

(5) *Bi-cristal diameter.* The bi-cristal diameter is a measurement of the width of the pelvic girdle. It represents the maximal distance between the external margins of the iliac crests. The bi-cristal diameter proved to be one of the most constant, most reliable (in the statistical sense) anthropometric characteristics of the individual.

(6) *Bi-trochanteric diameter.* The bi-trochanteric diameter was determined as the maximal distance between the external surfaces of the trochanters. In some individuals considerable pressure had to be exerted on the points of the calipers in order to minimize the error of including the width of the overlying tissues in the bi-trochanteric diameter.

(7) *Transverse diameter of the thorax.* The breadth of chest was determined as the distance between mid-axillary lines at the level of the nipples, at normal expiration.

(8) *Anteroposterior diameter of the thorax.* The depth of chest was determined as the distance from the mid-sternal line at the level of the nipples to the vertebral column, at normal expiration.

(9) *Circumference of the thorax.* The chest circumference was measured in the horizontal plane passing through the nipples, at normal expiration.

(10) *Circumference of the abdomen.* The measurement was made in the horizontal plane at the level of the navel, in normal expiration. Attention was paid to avoiding either of two errors: slipping of the steel tape and excessive tension in the tape.

(11) *Circumference of the upper arm.* The arm girth was obtained as the maximum circumference of the right upper arm, below the insertion of the deltoid muscle. The arm was hanging free alongside the body. The plane of the circle formed by the tape was perpendicular to the long axis of the arm. The level at which the girth was measured during the control period was determined in terms of the distance of the point on the dorsal side of the arm from the elbow, with the forearm flexed at 90° . This distance was recorded, and the level at which the measurements were made was kept constant throughout the experiment.

(12) *Circumference of the thigh.* The subjects sat on the edge of a standard chair, with the legs flexed at 90° . The girth of the right thigh was measured at the halfway point between the outer surface of the patella and the hip bone; the distance from the knee was recorded and marked with a soft pencil on the ventral side of the thigh every time the measurement was made.

(13) *Circumference of the calf.* The leg was relaxed, the position of the subject being the same as for measurement of the thigh (item 12). The circumference of the leg was determined as the maximum girth of the calf, again at the plane perpendicular to the long axis of the leg.

(14) *Weight.* The nude subjects were weighed daily, before breakfast but after emptying the bladder, on a beam balance. The weights were read to the nearest $\frac{1}{4}$ lb.

PHOTOGRAPHIC MEASUREMENTS

The measurements on the photographs were made by using Sheldon's technique (Sheldon *et al.*, 1940, pp. 54-57). The dimensions measured were indicated in Figure 1 in Lasker (1947, p. 326) and were defined as follows:

(1) *Upper facial breadth.* The distance between the junctions of the pinnae of the ears and the skin lines of the head. As in the set of pictures used for photometric analysis, the head has been blocked out in frontal and lateral views to prevent identification of individuals; the measurement was taken in the dorsal view.

(2) *Lower facial breadth.* The distance between the intersections of the lines of the ear lobules and the skin lines of the head; dorsal view.

(3) *Neck depth.* The minimum anteroposterior diameter of the neck; lateral view. In some cases this line had been obscured in blocking out the head and the measurement was omitted.

(4) *Neck breadth.* The shortest transverse diameter of the neck; taken in the frontal view, in most cases, as well as in the dorsal view. The mean difference between the values obtained by the two methods, frontal and dorsal, is nil (0.0 ± 0.3 per cent).

(5) *Upper trunk breadth.* The distance between the uppermost visible points of the posterior axillary folds; dorsal view.

(6) *Upper trunk depth.* The horizontal anteroposterior diameter taken midway between the level of the center of the nipple and the most anterior projection of the sternoclavicular junction; lateral view. The conical shape of the thorax and the complex method of locating the landmarks made it difficult to take this measurement accurately.

(7) *Waist breadth*. The minimum transverse diameter of the waist; dorsal view.

(8) *Upper arm thickness*. The anteroposterior diameter of the upper arm at the mid-point between the fold at the cubital fossa and the tip of the greater tuberosity of the humerus; lateral view.

(9) *Forearm thickness*. The forearm thickness at the cubital fold; lateral view.

(10) *Wrist thickness*. The diameter of the forearm in a plane two inches from the most anterior projection of the radius. The two inches were approximated by laying off the width of one of the background squares (5 cm.) with the calipers; lateral view.

(11) *Waist depth*. The minimum horizontal diameter of the waist; lateral view.

(12) *Hip breadth*. The maximum horizontal transverse diameter, usually found over the greater trochanters; dorsal view.

(13) *Pelvic depth*. The horizontal anteroposterior diameter taken at the level of the superior margin of the symphysis pubis; lateral view.

(14) *Upper thigh depth*. The horizontal anteroposterior diameter taken at the level of the center of the gluteal fold; lateral view.

(15) *Lower thigh depth*. The horizontal anteroposterior diameter at the level of the fossa immediately above the patella; lateral view.

(16) *Calf breadth*. The maximum transverse calf diameter in a plane at right angles to the long axis of the lower leg; dorsal view.

(17) *Ankle breadth*. The minimum transverse diameter of the ankle at right angles to the long axis of the lower leg; dorsal view.

(18) *Stature*. The vertical distance from the heels to the vertex of the head; dorsal view. Since the heels are partly obscured by the positioning blocks on the pedestal, and since the vertex is partially obscured by hair, it is necessary to approximate these points by interpolation.

A pair of vernier calipers reading to 0.1 mm. was used. All measurements were taken twice, at different times. When the two measurements were not in close agreement, the measurement was repeated several more times. The range of difference between the two trials on the same photograph exceeded 0.6 mm. only for stature and upper trunk depth, and in no case was there a difference of as much as one millimeter. The standard deviations of the repeated determinations on the individual photographs for the various measurements range from 0.1 to 0.3 mm., which is 2 per cent or less for all measurements except wrist thickness, in which it is 3 per cent of the mean dimension. In one or two instances photographs were repeated on the same individual. In these cases the differences in the measured diameters were very small: they do not exceed 0.6 mm. in any instance, and they have a standard deviation of less than 0.25 mm.

CONSTITUTIONAL PHOTOSCOPY

Utilizing Sheldon's concepts, Hooton (1946, p. 764) defines "somatotyping" as rating the development of each of the three "structural components" of the body build—endomorphy, mesomorphy, and ectomorphy. From three to five

features are evaluated in each of the five principal regions of the body. The means of the separate ratings represent the regional somatotypes; the regional values are averaged to give the over-all somatotype rating.

For characterizing the body build of the subjects in the Minnesota Experiment on the basis of inspection of photographs, the following criteria were used by the Harvard Anthropometric Laboratory (Hooton, 1946, pp. 767-69):

Region I. HEAD AND NECK

<i>Endomorphy</i>	<i>Mesomorphy</i>	<i>Ectomorphy</i>
1. Little bony relief (pneumatic fullness); basketball head and face	Muscular, rugged, bony face; square or oblong tendency in head and face	Thin, fragile-boned face (often environmentally malformed); oval or hatchet face (but highly variable)
2. Chin-neck angle blunted	Sharp chin-neck angle; heavy, square chin	Pointed, or weak, light chin; sharp chin-neck angle
3. Pudgy features; often short, pointed nose with conical tip; thick, loose lips; tendency toward "suckling" lips	Heavy, broad nose of variable length; firm, large mouth	Delicate features, especially nose and mouth
4. Smooth, cylindrical neck (not necessarily short); Anteroposterior \doteq Transverse diameter	Heavily muscled, "bull" neck; pyramiding of trapezius; Anteroposterior less than Transverse	Thin, long neck, often inclined forward; Anteroposterior \doteq Transverse
5. Soft-padded clavicles	Heavy, strong clavicles	Delicate, sharp clavicles (sternal ends often dropped); marked clavicular hollows

Region II. THORACIC TRUNK

<i>Endomorphy</i>	<i>Mesomorphy</i>	<i>Ectomorphy</i>
1. Back: smooth, no muscular relief	Back: rugged, high muscular relief	Sharp bony relief; little muscular relief; scapulae often winged
2. Back: markedly broad; faint and sometimes reversed taper (even absent)	Back: markedly broad; sharp taper (disappears in 6's and over)	Narrow back; slight to medium but highly variable taper
3. Side: deep, puffy chest; lower depth exceeds upper; elastic (high expansibility, big lift)	Deep, muscular chest; lower depth \doteq upper	Flat, shallow, non-muscular chest
4. Side: abdomen predominant over thorax; both deep and wide	Thorax predominant over muscle-controlled abdomen	Thorax either predominant over small, compressed abdomen or subordinated to small convexity (indicative of mesopenia)
5. Front: high, wide, faint rib angle; relatively short chest cage	Heavy, well-muscled ribs; moderate rib angle; chest long relative to abdomen	Skinny ribs; sharp rib angle; total trunk short, but thorax relatively medium or long

Region III. ARMS, SHOULDERS, AND HANDS

<i>Endomorphy</i>	<i>Mesomorphy</i>	<i>Ectomorphy</i>
1. Shoulders square, high; fat-blanketed	Shoulders prominent, broad, muscular, often sloping	Shoulders narrow, bony, thin; height variable; often rounded
2. Proximal hamming; thickness proximal exceeds distal segment; little muscle relief; smooth, tapering forearms; small rounded wrists; Anteroposterior \equiv Transverse	Rugged deltoid, triceps, biceps; massive muscular forearm nearly as thick as upper arm; heavy, square, bony, muscular wrists; superficial veins marked	Stringy, thready muscles; low relief; long, weak, bony forearm; thin, fragile, bony wrists
3. Tendency toward short-fingered, pudgy, small-boned hands (marked dysplasias)	Massive, heavily muscled, square bony hands	Thin, narrow hands; slender digits

Region IV. ABDOMINAL TRUNK

<i>Endomorphy</i>	<i>Mesomorphy</i>	<i>Ectomorphy</i>
1. Full, large inflated abdomen; Anteroposterior \equiv Transverse	Compact, well-muscled abdomen; frequent rectus relief; Anteroposterior less than Transverse	Underdeveloped, non-muscular, small abdomen; frequently with slumped convexity or otherwise variable
2. Broad, high, indistinct waist	Sharp, low, well-muscled waist of variable breadth	Small, fragile, non-muscled waist
3. Broad pelvis with fat pads and greatest breadth above crests (when well nourished)	Heavy, bony pelvis, muscle markings over Poupart's ligament	Narrow, sharp-boned pelvis; conspicuous anterior iliac spines
4. High, flat lumbar curve	Sharp, low lumbar curve	High, deep lumbar curve
5. Inflated, soft, large buttocks; angle with gluteal fold partly obliterated in lateral view	Muscular, laterally dimpled buttocks (due to endopenia); sharp angle with gluteal fold	Flat, thin, non-muscular buttocks (micropygy due to mesopenia)

Region V. LEGS AND FEET

<i>Endomorphy</i>	<i>Mesomorphy</i>	<i>Ectomorphy</i>
1. Soft, pneumatic fullness; proximal predominance and hamming of thighs	Thick, ruggedly muscled, heavy-boned legs; solid, even development of segments	Thin, non-muscular, bony legs with delicate, elongated, distal segments
2. Approximation of thighs when heels together (knock-knees in mesopenia); predominance of outer calf curve (weakness of inner); small, rounded ankles	Prominent muscular relief of massive thighs; conspicuous inner calf curve; gastrocnemius calf shadow; ankles massive, bony	Weak muscling of thighs; marked interspace; relatively little muscling or curvature of calves; fragile, sharp-boned, thin ankles
3. Small-boned, pudgy feet; toes usually short but frequently dysplasias	Large, heavy, bony feet; toe length variable	Long, thin, delicate feet (dysplasias frequent)

Body Density and Calculated Body Fat

Specific gravity is defined as the weight of a substance compared with the weight of an equal volume of another substance. Using water as the standard,

$$\text{Specific gravity} = \frac{\text{Body weight in air}}{\text{Volume of water displaced}}$$

The volume for calculations of the specific gravity of the body in the Minnesota Experiment was obtained indirectly by weighing the men under water. According to the Archimedean principle, a body immersed in a fluid loses in weight by an amount equal to that of the fluid displaced. Accordingly: Volume = Weight in air — Weight in water. The formula for specific gravity can be then written:

$$\text{Specific gravity} = \frac{\text{Body weight in air}}{\text{Body weight in air} - \text{Body weight in water}}$$

The men were weighed under water in a deep pool the temperature of which was maintained at about 78° F. To secure full submersion, the subjects were attached to a lead sinker. Support was provided by a rubber sling, placed under the subject's arms and fastened by a cord to one end of a balance beam. The beam had a fulcrum at the edge of the pool, one arm extending above the water and the other arm attached to a counterweight placed on the platform of a balance. The body weight under water was then obtained as the difference between the "control" reading on the balance, representing the counterweight corrected for the sling and the sinker, and the "weighing" reading.

The measurements were made under conditions of maximal expiration; the subjects were thoroughly trained in the technique during the control period and repeated measurements yielded consistent results. Even at maximal expiration some air remains in the lungs and the air passages ("residual air"), which increases the buoyancy of the body. The value for body weight in water must be corrected for this factor. It was not technically feasible to make determinations of residual air simultaneously with underwater weighing. Efforts to estimate individual values for the residual air from anthropometric or radiological measurements and vital capacity determinations have not been very satisfactory. Hurtado and Fray (1933) obtained in a series of 50 males a correlation coefficient of 0.457 between vital capacity and residual air, which is too low to allow prediction of the latter variable from the value for vital capacity. Attempts to predict the residual air volume from radiological measurements are even less satisfactory, the correlation with the chest volume at maximal expiration being $r = 0.383$ and the correlation with lung fields $r = 0.371$. The two anthropometric dimensions measured had no predictive value: for the chest circumference $r = -0.07$; for the external chest volume $r = 0.11$.

We have computed coefficients of correlation between various anthropometric characteristics and the residual air for a series of measurements on 25 professional football players, published by Welham and Behnke (1942). The rank-correlation method was used. The values in Table 436 indicate the absence of any correlation which would be high enough for an accurate estimation of

TABLE 436

COEFFICIENTS OF RANK-CORRELATION BETWEEN ANTHROPOMETRIC CHARACTERISTICS AND RESIDUAL AIR. *N* = 25 athletes. (Data from Welham and Behnke, 1942.)

	Residu- al Air	Vital Capacity	Lateral Chest Diameter	Height	Chest Circum- ference	Weight	Anteroposterior Chest Diameter
Residual air560	.348	.269	.252	.083	— .013
Vital capacity560		.200	.360	.218	.203	.128
Lateral chest diameter348	.200		.164	.725	.435	.218
Height269	.360	.164		.323	.544	.313
Chest circumference	.252	.218	.725	.323		.777	.668
Weight083	.203	.435	.544	.777		.813
Anteroposterior chest diameter. .	— .013	.128	.218	.313	.668	.813	

residual air. Even after combining the independent variables into a multiple regression equation, the error in predicting the dependent variable (residual air) would remain large.

Since we were concerned with the absolute values primarily for the group as a whole, it was feasible to make an estimate of the average residual air volume which can be regarded as reasonably accurate. The value of 1450 cc. (equivalent to 3.21 lbs. of displaced water) has been used as a correction in computing the mean values for specific gravity. Behnke, Feen, and Welham (1942) reported that with groups of 20 or more men the use of this estimated average figure for residual pulmonary volume will not introduce an error greater than ± 0.003 in the computation of specific gravity.

In a group of 25 professional football players Welham and Behnke (1942) found a mean value for residual air of 1.444 liters, with a relatively narrow range from 1.187 liters to 1.930 liters. The mean value computed from figures given by Welham and Behnke (1942) for 75 Navy men was 1.509 liters. In another group of 99 men, presumably also Navy personnel, the mean obtained on the basis of data by Behnke, Feen, and Welham (1942) was 1.409, with a range from 0.706 to 2.650.

Similar figures were obtained by Hurtado and Boller (1933). They collected from the literature since 1918 determinations on male subjects of pulmonary capacity and its subdivisions, including residual air volume. The mean value for residual air was 1.50 liters, standard deviation 0.33 liters, range from 0.87 to 2.48 liters. These authors report also their own findings, obtained on 50 normal men varying in age from 18 to 30 years, with a mean age of 23 years. The anthropometric characteristics were similar to those of our experimental group, although the chest circumference varied within wider limits than in our sample. The following values were obtained for residual air: mean, 1.36 liters; standard deviation, 0.38 liters; range from 0.81 to 2.16 liters.

All the determinations mentioned so far were made "in air," not under water. What is the effect, if any, of immersion in water on the magnitude of the residual air volume? Hamilton and Mayo (1944) reported that vital capacity is decreased

by some 300 cc. when the body is immersed in water to the nipple line, as compared with the value obtained for the subjects standing, the averages for 10 subjects being 4548 cc. and 4863 cc., respectively. Placing diastolic pressure cuffs around the bases of the upper and lower extremities, which results in a decrease of venous return, increased the average value of vital capacity in water to 4721 cc. The authors inferred that immersion increases the load of blood in the lungs and decreases the space available for air. This factor would tend to affect the residual air volume in the same way. On the other hand, the forced expiration against the pressure of water may be less effective, leaving a larger part of air in the lungs. The two factors, acting simultaneously and in opposite directions, may tend to cancel out.

The effect of submersion was investigated experimentally by Brožek, Henschel, and Keys (1949). Determinations of the volume of residual air were made under ordinary room conditions and during complete submersion. Nine normal young men served as subjects. The average values, in cc., of the residual air volume, were 1590 and 1472 during the first trial and 1566 and 1426 during the second trial made a week later. There were large individual differences in the responses to submersion and the over-all average decrement of 129 cc. did not quite reach the 5 per cent level of statistical significance. Even had the difference been significant statistically, the absolute value is not large enough to introduce an important error in the estimation of the fat content of the body.

In addition to its value per se, the specific gravity determination has been useful for estimating the changes in the amount of body fat. The value for specific gravity of the human body — including tissue, bone, and essential lipids, and excluding excess fat — was estimated by Behnke (1941-42) as 1.099; the addition of excess adipose tissue amounting to 10, 20, and 33.3 per cent of the total body weight was calculated to yield 1.080, 1.062, and 1.036, respectively, as the values for specific gravity.

Experimental validation of the use of specific gravity for estimating the fat content of the body has been carried out by Rathbun and Pace (1945). Guinea pigs exhibiting a wide range of variations in weight and fat content were used. The specific gravity of the eviscerated animals and of the viscera was determined by the water displacement method, the fat content was obtained by petroleum ether extraction, and an equation was derived for prediction of the fat content of the body from the specific gravity. On the basis of these empirical data and some theoretical considerations, a provisional equation was formulated for the estimation of human body fat:

$$\% \text{ fat} = 100 \left(\frac{5.548}{\text{specific gravity}} - 5.044 \right)$$

Rathbun and Pace (1945, p. 674), constructed a table relating values of specific gravity from 1.002 to 1.100 directly to fat expressed as percentage of the body weight (range, 49.3 per cent to 0.0 per cent). This table has been used in our calculations.

The technique is reliable, in the statistical sense, yielding under standard conditions relatively constant values for the individuals. When the specific grav-

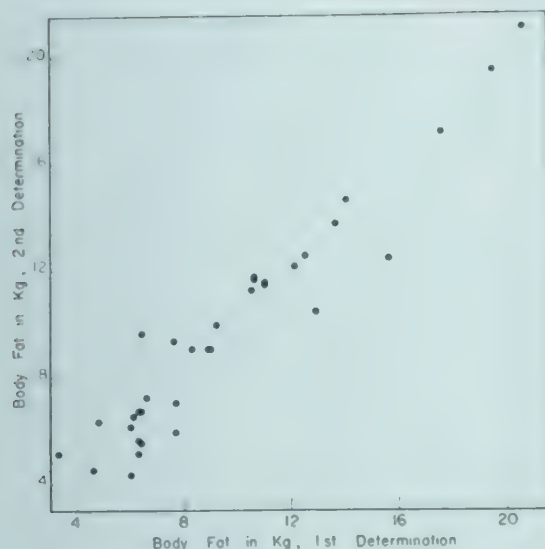


FIGURE 145. DUPLICATE BODY FAT DETERMINATIONS MADE IN THE CONTROL PERIOD 4 DAYS APART (Minnesota Experiment).

ity determinations were repeated within a period of from three days to a week, the values for the specific gravity of the body differed in 90 per cent of the cases by less than ± 0.003 units. Each determination was based on two measurements of body weight in water. The coefficient of correlation between the two sets of body fat determinations was $+0.95$ ($N = 35$) (see Figure 145).

The validity of the method depends on the correctness of the assumptions on which the equation for translating the specific gravity into percentage of body fat has been based. Morales *et al.* (1945) considered fat the only major body component whose relative value exhibits appreciable interindividual variations; the proportions of other body components were assumed to be relatively constant. In a subsequent paper the statement was made more precise and limited: "Perhaps the only whole body component, and certainly by far the chief one, which may fluctuate in the so-called 'normal' population is fat" (Pace and Rathbun, 1945).

In our subjects the semi-starvation regimen was accompanied by a complex set of changes including, in addition to the decrease of body fat: (1) a decrease of muscle mass; (2) an increase in hydration indicated by a marked, visible edema in a large number of men and a relative increase in the volume of extracellular fluid in all subjects; and (3) an increase in the relative mass of bony tissues.

Thus the proportion of the constituent components differs from the "normal" conditions assumed in the development of the formula for estimation of body fat on the basis of specific gravity.

Fortunately, there is a tendency for the errors to cancel out. Thus the increased hydration would tend to decrease the body density and increase the apparent percentage of body fat. A relative decrease of the active tissue, having approximately the same specific gravity as the extracellular fluid, tends to balance out the increase in hydration. The question of compensating for the shifts in the relative proportions of different parts of the body is discussed in greater detail in Chapter 15.

The Quantitative Estimation of Bone Density

(By Pauline Beery Mack, Hughes Daniel Trapp,* and Walter N. Brown, Jr., from the Ellen H. Richards Institute of Pennsylvania State College.)

In the past the common method for the appraisal of bone density from roentgenograms has been by subjective impressions. An attempt was made by Mack in 1927, by Stein (1937), and by Sanders (1937) to achieve a somewhat objective basis through the use of a stationary beam of light passing through a certain area of X-ray film, also stationary, onto a photronic cell. The use of a static technique of this character, however, is attended by large errors, because the identical anatomical position in the skeletal X-ray of the bone of different animals or human subjects, or of the same subject at successive times, cannot be located accurately.

In 1929, Mack adapted a photographic tracing densitometer, operating on the photoelectric principle, to skeletal roentgenograms. Although reproducibility was poor with this original instrument, particularly with any but large bones, its use demonstrated that the principle of making tracings from one landmark to another by means of a stationary beam of light and a moving film eliminated the basic difficulties of the static technique.

In 1936, a Type B Moll recording microphotometer was obtained; the instrument was designed for making tracings of spectral photographic plates. Various features of the instrument rendered it unfit in its original condition for the purpose of tracing X-ray material of human beings and experimental animals. Through the technical assistance of Ernest Axman and Warren Mack, the instrument was modified in many respects. After 2½ years of successful use of this device with roentgenograms of human subjects, Mack *et al.* (1939) described the method as then developed. From 1942 to 1945 the microphotometer and the general method of its use underwent numerous further refinements by Dr. Trapp. These have increased its precision and widened the range of bones which could be evaluated; the method now is applicable to anything from the mouse femur to the skull of man.

The accuracy of the microphotometric scanning procedure for making photographic tracings of animal and human bones rests upon the validity of two assumptions: (1) that the density of the image on a roentgenogram is related in a direct manner to the density of the mineral of the bone upon which the roentgenogram is based; and (2) that, through the use of a calibration ladder or stile placed on the film at the time when the exposure is made, corrections can be applied for unavoidable variations in exposure and development technique. This reference material must be as nearly homogeneous as possible in both density and chemical composition. It should, as closely as possible, resemble in chemical composition and X-ray absorption spectrum the object the density of which is being evaluated.

Knowledge of the atomic composition of the object under examination is necessary for the assessment of mineral density from roentgenograms. Fresh human bones are made up of from something like 20 to 32 per cent minerals, the average for the entire skeleton analyzed by Volkman (1873) being 22.11 per cent

* Died October 20, 1945.

ash and that reported by Mitchell *et al.* (1945) being 28.91 per cent ash. But these minerals account for more than 90 per cent of the total X-ray absorption, as will be seen by comparing the relative atomic absorption coefficients at a typical wave length, $\lambda = 0.25 \text{ \AA.}$, for the major elements in bone: calcium 55, phosphorus 17.5, oxygen 1.41, carbon 0.45. The calcium content of bone ash averages from 35 to 40 per cent and the phosphorus content from 15 to 18 per cent (cf. Mitchell *et al.*, 1945).

It is clear that the X-ray absorption by bone is due principally to calcium and that for bones having a chemical composition within the range of variation indicated in the literature, the X-ray absorption of bone is nearly proportional to the number of calcium atoms present. The next most important element in the absorption of X-rays, phosphorus, accounts for perhaps 10 per cent of the total absorption.

If continuous spectrum X-rays are used in the evaluation of calcium density, a procedure must be found to take account of the variation of spectrum shape and energy absorption rate with penetration, to correct for the variation of film sensitivity with X-ray wave length, and to correct for the many other variations which inevitably take place in any photographic exposure and development process. These obstacles can be overcome by exposing on the same film as the examined bone a multiple-step ivory ladder or wedge of accurately known physical dimensions and chemical composition. The chemical composition of ivory, although not identical with that of bone, is remarkably equivalent in terms of X-ray absorption.

The "ideal" procedure for converting roentgenographic density to calcium density of the bone is long and tedious. An approximate roentgenographic evaluation technique which involves considerably less labor has been in use for a number of years in the laboratories of the Ellen H. Richards Institute and has been found entirely adequate for nutritional studies. This standardized technique leads to a Mineral Index very nearly proportional to the calcium density of the examined bone.

In making roentgenograms, X-ray film of the double-coated acetate base type is used. It is necessary to employ a plumb bob for locating the X-ray tube with respect to a standard position over the part of the body being filmed and to use a highly standardized technique of exposure and development. The following are the essential steps involved in taking a roentgenogram and in making and evaluating a photographic tracing by the microphotometric method:

(1) The X-ray film is exposed in a standard position for any part of the body which is to be used, employing an exposure energy suitable to the thickness of the object examined. This is called FILM A. A second film, taken at right angles to the first, is called FILM B. A calibrated ladder is used in a standard position on each film. After exposure, the X-ray films are developed, fixed, and washed under as nearly uniform conditions as can be achieved.

(2) A contact positive is made from FILM A, which is the film from which microphotometric tracings are made. In order to secure maximum reproducibility of data, systematic procedures have been evolved for establishing the exact position of the axes of the bone image along which the densitometer traces are

made. The positive is used for locating tracing paths (to be followed by the beam of light in the microphotometer) without damage to the X-ray film itself. The ends of the tracing paths then are defined on the film by means of small ink dots outside the bone area proper.

(3) Tracings are made through the predetermined path or paths on the film and through the ivory ladder image.

(4) Each bone tracing is divided into a finite number of segments in accordance with a standardized procedure.

(5) The area under each segment is measured by means of a Carodi planimeter.

(6) The heights of the definable steps in the ladder tracing are measured.

(7) An interpolation formula is used to correct the area under each segment of the bone tracing for exposure and development deviations in terms of observed height of trace and measured physical thickness of the adjacent steps on the standardization ladder. The corrected segment areas then are added to obtain a corrected area value for the entire trace.

(8) Linear measurements are made both on FILM A and on FILM B, which enable calculations to be made of the cross-sectional area of bone through which the X-rays that passed through the tracing path have penetrated.

(9) The corrected area under the trace is divided by the volume of bone through which the X-ray beam has penetrated (calculated area in Paragraph 8 times one millimeter effective beam width). This gives the mineral index of the bone in terms of ivory equivalent thickness per unit volume of bone in this particular position.

The microphotometer functions essentially as follows: A stationary beam of light passes through a lens condensing system, thence through the roentgenogram, and finally onto a photronic cell; this cell is in a light-tight box. The image of the brightly illuminated X-ray area is focused on the plane of an adjusting slit on the near side of the photronic cell, and thence onto the cell. The latter is connected directly with a sensitive galvanometer. The film being scanned is mounted on a carriage which moves at a constant velocity of approximately 0.01 inches per second past the scanning beam. The carriage is moved by the same synchronous motor which turns the drum on which the photographic recording paper is placed. This assures a constant ratio between the linear velocities of the carriage through the scanning beam and the surface of the recording paper. The instrument thereby is a recording densitometer. It is essential that the intensity of the scanning lamp be controlled as closely as possible. It is operated in a direct current circuit designed to provide adequate control of the voltage across the lamp.

The microphotometer must be operated in a darkroom. It is convenient to use a second darkroom for the processing of the photographic paper. The drum accommodates recording paper up to 12×40 centimeters. Light from the galvanometer mirror is admitted through a slot and brought to a focus on the surface of the paper by a cylindrical lens mounted so that its axis is perpendicular to the long dimension of the paper.

The height of the tracing is proportional to the light transmitted through the

corresponding point on the X-ray film, which in turn is a substantially linear function of the amount of mineral matter through which the X-ray radiation has passed during exposure of the film. The area of the cross section (estimated from two exterior measurements of the bone and of the cavity) multiplied by the width of the path on the roentgenogram image scanned by the densitometer gives a close approximation of the volume of bone for which the average mineral index is evaluated. No bone positions are traced except those for which cadaver bones have been studied to assist in estimating bone shapes in directions other than the two for which X-rays may be taken.

Experimental checks on the reproducibility of the technique have been carried out by evaluating the mineral index from a number of roentgenograms prepared at the same time for the same bone under varying conditions of exposure and development. There is a wide variation with development time of the mineral index value that might be computed on the basis of film density alone. The corrected mineral index, obtained by correcting the density of the bone image for each film on the basis of the photographic traces made of the ladder step images on the same film, does not vary greatly over the wide range of development conditions.

Biochemical Analyses

DETERMINATION OF RIBOFLAVIN IN THE URINE

The method used was a modification of the Conner and Straub fluorometric procedure (1941). A urine blank in which the riboflavin was destroyed by irradiation was put through the entire procedure for each sample. The riboflavin content was then corrected for the residual fluorescence of the blanks. Instead of duplicates, two aliquots of the same sample, differing in volume by a factor of two, were run; the difference between these results had to be less than 10 per cent of the mean before the values were accepted.

DETERMINATION OF PYRAMIN IN URINE

Pyrimin is the name that has been given to the pyrimidine-like portion of the thiamine molecule that is excreted in the urine. The procedure as developed by Schultz, Atkin, and Frey (1942) was for the determination of thiamine. In their method pyrimin appears as the blank which is subtracted from the value for the total fermentation in order to get the corrected thiamine. Their procedure with the following modifications was used for the determination of pyrimin:

(1) *Sulfiting*. By means of a Krogh-Keys pipette (Keys, 1937), 30 cc. of urine were put in a test tube calibrated to contain 35 and 50 cc. To this were added 1 cc. of a 15 per cent sodium sulfite solution (anhydrous) and 3 drops of 9 N sodium hydroxide. The samples were mixed by inverting the tube several times; a rubber stopper was inserted in the tube during this operation. The solution was adjusted to a pH of 5.3 to 6.8 with bromocresol blue as an outside indicator. The tube was covered with a small beaker and placed in a bath of boiling water. Thirty minutes after the bath again started to boil, the tube was removed and to it was immediately added 1.1 cc. of 3 per cent hydrogen peroxide. The tube was shaken during the addition of the hydrogen peroxide. As soon as the solution had cooled to room temperature, 3 drops of 9 N sodium hydroxide were

added to the tube. The remaining sulfite was destroyed by means of additional hydrogen peroxide. The amount of peroxide necessary for this was controlled by means of a starch-iodide indicator, which was made by mixing in a depression of a spot plate 1 drop of 6 per cent potassium iodide, 1 drop of 50 per cent sulfuric acid, and 1 drop of 1 per cent starch suspension. After standing a few minutes this indicator developed a pale rose color, which became dark blue on the addition of peroxide and was decolorized in the presence of sulfite. By means of 3 per cent hydrogen peroxide (occasionally 1 per cent hydrogen peroxide or 5 per cent sodium sulfite was required) the contents of the tube were adjusted so as to produce no change in color when one drop was added to a fresh starch-iodide mixture. The pH was adjusted to 7.0 and the volume brought up to 50 cc. with distilled water. When the pyrimin excretion was expected to be very low, the volume was brought up to the 35 cc. mark.

The distilled water should be checked against specially prepared double-distilled water in order to ensure the absence of chlorine or other substances that might affect the yeast fermentation.

The volume of the sulfited urine required for the fermentation test was calculated from the urine volume on the assumption that a normal male adult on an ordinary diet would excrete 200 micrograms of pyrimin per day (expressed as 2-methyl-4-amino-5-ethoxymethylpyrimidine hydrochloride). To each fermentation bottle was added approximately 0.5 microgram of pyrimin.

All samples were sulfited within 3 days after the urine was collected. Sulfited samples stored in the refrigerator are stable for 2 weeks.

(2) *Fermentation procedure.* The solutions were the same as those described by Schultz, Atkin, and Frey (1942) except that 4 mg. of pyridoxine were added to each liter of solution A. Both solutions A and B were made up and autoclaved in 4-liter pyrex bottles each equipped with a siphon, the delivery end of which was covered with a test tube except when actually in use. The air entering the bottle passed through a calcium chloride tube filled with sterile cotton. These solutions, thus kept, are stable for over 6 months. The yeast suspension was made by adding 25.0 gm. of Fleischmann baker's yeast to 500 cc. of distilled water. This solution was stirred mechanically while the yeast was added to the media. Krogh-Keys pipettes were used in adding solutions A and B and the yeast suspension to the fermentation flasks.

The yeast suspension was added to successive flasks at exactly 10-second intervals. The "zero time" of the fermentation reaction was calculated from the moment the yeast suspension was added to the first fermentation vessel. The initial gas volume readings were not made until 20 minutes after the yeast was added to the first vessel. Each buret was read at 10-second intervals in the same order that the yeast suspension was added. When two people worked together, the yeast could be added to 24 fermentation vessels, the vessels connected to the burets, and the shaking started 3.5 ± 0.25 minutes after the addition of the first yeast suspension.

(3) *Calculation of results.* Routinely a blank (zero microgram standard) and five standards (such as 0.2, 0.4, 0.6, 0.8, and 1.0 micrograms of 2-methyl-4-amino-5-ethoxymethylpyrimidine hydrochloride) were included in each ferment-

tation run. All gas volumes were corrected to standard conditions of temperature and pressure. In order to secure the gas evolution elicited by the added pyrimidine, the gas produced by the blank fermentation vessel was subtracted from each of the standards. These differences were plotted against the standards to get the standard response curve. Such a curve was very constant over a period of months and served as a check on the correctness of the daily blank. The concentration of pyramin in the unknowns was calculated from the standard curve and expressed in terms of 2-methyl-4-amino-5-ethoxymethylpyrimidine hydrochloride.

NITROGEN BALANCES

For a 3-day period each of the men in the nitrogen balance group was given the standard quantities of food on the menu for that day. Just before breakfast on the first day each man received a capsule containing carmine. This was repeated 3 days later. The carmine was used to delineate the stool samples. All stool and urine samples during the interval were saved.

Each stool specimen was weighed and then homogenized in a Waring blender with a measured amount of 0.1 N hydrochloric acid. Enough acid was added to permit a uniform mixture, the consistency of very heavy cream. The weight of the acid was added to the weight of the stool, and the figure thus secured was used in calculating the nitrogen content. A 10 per cent sample of individual specimens obtained during a particular period was put into an ointment jar. At the end of the period, these were blended and stored in the deep freeze until ready for analysis. A 5 gm. sample was digested with a sulfuric-phosphoric acid mixture with selenized Hangar granules as the catalyst. After the mixture clarified, the digestion was continued for another $\frac{3}{4}$ hour. The ammonia was distilled into a boric acid solution and back-titrated with standardized acid. From this result, the nitrogen in the 3-day stool samples was calculated.

The urine was preserved with glacial acetic acid (3 cc.) and toluol (5 cc.). The 3-day urine samples were pooled and an aliquot saved for analysis. The amount used in the digestion depended upon the size of the urine volume. It was digested and distilled the same as the stool specimen.

Complete dietary samples were saved. These were weighed and mixed after 0.5 per cent of Duponal had been added to improve and facilitate the preparation of the sample (Mickelsen *et al.*, 1947). The mixture was put through a power-driven meat grinder, then mixed in a kitchen mixer, after which a sample was homogenized in the Waring blender. Two-inch-long cartridges made out of Parafilm[°] were filled to within a half-inch of the top. The weight of the sample added to the cartridge was secured by the difference in weight before and after filling. The top edge of the Parafilm cartridge was pressed together and then sealed with a glass rod heated in a flame. Each sample was identified by means of a label stapled to the cartridge. All necessary data for the samples were recorded in a notebook. These cartridges were stored in the deep freeze until ready for analysis. Then the wax was easily removed from the sample, which was digested and distilled the same way as the stool specimen.

[°] Secured from the Standard Scientific Supply Company, 34 West Fourth Street, New York, New York.

BLOOD AND PLASMA VOLUME DETERMINATIONS

The blood and plasma volumes were determined by the dye dilution (T-1824) method of Gibson and Evans (1937) as adapted to the photoelectric colorimeter by Gibson and Evelyn (1938). In all cases Evans Blue (T-1824), lot Number 3878, from the Eastman Kodak Company was used.

All the glassware used in the preparation of the dye solutions was washed and rinsed in distilled water, treated with dichromate cleaning solution, rinsed in 5 changes of tap water, 8 changes of single distilled water, and 6 changes of double glass-distilled water, and thoroughly dried in an oven at 98° C. The dye solution was made up in sterile isotonic saline to the approximate concentration of 1.0 to 1.5 mg. per cc. The dye solution was filtered through a sintered glass filter and transferred to ampoules in an ultraviolet light chamber. The filled ampoules were sealed and autoclaved immediately for 30 minutes at 15 pounds pressure.

A sample of the dye solution was removed after the filtration for the gravimetric determination of the precise dye concentration. The combined plasma volume and extracellular fluid injection solution was prepared by adding 50 mg. per cc. of analytical grade sodium thiocyanate to the filtered dye solution before the solution was placed in the ampoules.

All plasma and extracellular fluid volume determinations were made in the mornings, 12 to 16 hours post-prandial. The subjects avoided any but mild activity for 2 hours before the tests, and a half-hour of supine rest immediately preceded the injection of the dye. Ten cc. of the dye solution were injected into the antecubital vein in 60 seconds. A dye-free blood sample was taken before the injection, and dyed samples were taken at 20, 30, and 40 minutes after the injection of the dye. When extracellular fluid volume was also determined, post-injection blood samples were also taken at 10 and 50 minutes. All the blood samples were drawn from the antecubital veins with a minimum of stasis into oiled syringes and transferred immediately into paraffined tubes containing about 5 mg. of dry heparin to prevent coagulation.

Hematocrit values were determined on the basal and the 20-minute dyed samples. The hematocrit tubes were centrifuged at 3000 r.p.m. for 60 minutes, and the average value for the two tubes was used in the calculations of the total blood volume.

The blood samples were centrifuged at 2500 r.p.m. for 30 minutes, and exactly 6 cc. of the plasma from each sample were carefully transferred to colorimeter tubes. With the stop at 6 cc. the plasma samples were read with filters 620 M and 540 M in an Evelyn Colorimeter set at 100 with a water blank. The L values for the colorimeter readings were obtained. The corrected L values were plotted against time, and the theoretical L value for zero time was obtained by extrapolating the dye concentration curve back to zero time.

The use of an average K value determined from a number of plasmas in the calculation of plasma volume often leads to an error in the calculations. A K value was determined for each dye-free plasma sample by adding a known amount of the dye to the dye-free sample; 0.2 cc. of an Evans Blue solution containing exactly 0.1200 gm. per liter was added to 6 cc. of the dye-free sample.

This gave a dye concentration in the dyed dye-free sample that was about the same as that in the dyed samples. The plasma and total blood volumes were calculated from the formulas:

$$\text{Plasma volume in cc.} = \frac{\text{mg. dye injected}}{\text{extrapolated L 620}} \times K$$

$$\text{Total blood volume in cc.} = \frac{\text{plasma volume}}{100 - \text{per cent cells}} \times 100$$

DETERMINATION OF EXTRACELLULAR FLUID SPACE

Injection solution. The injection solution in each 10 cc. contained from 10 to 15 mg. of T-1824 (Evans Blue dye) and 500 mg. of sodium thiocyanate (A.R. grade). The concentration of thiocyanate in each batch of solution was checked by occasional analysis.

Collection of samples. The blood volume and the extracellular fluid determinations were made simultaneously. Besides the regular blood samples drawn for the plasma volume determination (basal, 20, 30, and 40 minutes after the injection of the dye), additional samples were secured at 10, 50, and in some cases 90 and 120 minutes after the injection of the dye. In the latter cases, only 6 cc. of blood were drawn. The blood was collected in oiled syringes and carefully emptied into paraffined centrifuge tubes. Heparin was used as the anticoagulant throughout, since both oxalate and citrates interfered with the colorimetric determination of thiocyanate. Numerous experiments showed that heparin had no influence on the thiocyanate analysis.

Method of analysis. As soon as the estimation of the dye (T-1824) in the plasma was made, the thiocyanate analysis was started. The procedure as described by Bowler (1944) was used except that the intensity of the colored solution was determined by means of an Evelyn photoelectric colorimeter which contained a filter with a maximum transmission at 440 m μ . The presence of the dye (T-1824) was shown to produce no influence on the thiocyanate determination. Both the plasma and the trichloroacetic acid filtrate may be left in the refrigerator overnight without producing any change in the results.

Calculation of extracellular fluid space. The thiocyanate concentration in the various plasma samples was plotted against time. In most cases there was a sharp inflection in the curve 20 to 40 minutes after the injection of the dye solution. The plasma concentration at that time was used as the equilibrium value (Crandall and Anderson, 1934). When there was no discernible break in the curve for a particular subject, the mean of the equilibrium times for the other subjects in the same group was arbitrarily used. This equilibrium value was then corrected for the thiocyanate-like material in the control sample to get the corrected equilibrium value. The weight of thiocyanate injected (in mg.) was divided by the corrected equilibrium plasma concentration (expressed as mg. per liter) to get the extracellular fluid space (in liters).

ELECTROPHORETIC ANALYSES

Blood samples of 25 to 40 cc. were drawn in the postabsorptive state from the resting subjects: oiled syringes were used, and none of the samples used

showed any trace of hemolysis. The blood was allowed to clot for at least 90 minutes and was then centrifuged. The separated serum was frozen and stored at -20° C. When the analysis was to be made, the serum was thawed, diluted with 2 parts of buffer solution, and dialyzed in collodion tubes for 3 to 5 days with frequent changes of the external buffer. A veronal buffer solution was used which had an ionic strength of 0.1 and a pH of 8.6.

The electrophoretic technique of Tiselius (1937) was used with the improvements suggested by Longworth (1939a, 1942). The scanning method of Longworth (1939b) was used, and the cells were of the single center section type (Longworth, 1940). Temperature was maintained at 2.0° C. during electrophoresis with a current strength of 12.5 milliamperes at 165 volts. The electrophoresis usually continued for about 200 minutes.

Photographic enlargements of the plates were made and the areas of the patterns were measured with a planimeter, using the general procedure and method of computation described by Tiselius and Kabot (1939) and discussed by Longworth (1939b). The electrophoretic spread of the various components of the serum was so controlled that a portion of the base line was visible on each side of the pattern. This permitted accurate construction of the base line under all the peaks. The albumin peak was measured only in the descending pattern, the ratio A/G being estimated from this pattern. The ascending and descending patterns were averaged in order to obtain the highest accuracy in the estimation of the globulins.

Total proteins were determined by the micro-Kjeldahl method of Keys (1940).

Semen Analysis

Collection of specimen. Semen specimens were collected, by masturbation, in clean, dry wide-mouthed bottles equipped with cork stoppers. The date and time of collection were noted on the bottles, and specimens were examined initially within three hours of collection.

Volume. The volume of a specimen was determined by drawing the entire specimen up into a special calibrated 10 cc. pipette. This same pipette was used to determine the relative viscosity.

Viscosity. The relative viscosity of the specimens was graded by the following methods. Using the same pipette throughout, the time required for the top of the column of semen to pass from the 8.0 cc. mark to the 9.0 cc. mark was recorded following volume determination. If this time was less than one second, the viscosity was graded 1 plus (1+). The grading of 4 plus (4+) was made if the time was greater than five seconds. Between one and five seconds the grades 2 plus (2+) and 3 plus (3+) were used. In the specimens whose volumes were less than 2.0 cc., the viscosity was graded less accurately by comparing the tenacity of the specimens as a stirring stick was withdrawn above the surface.

Turbidity. This determination was wholly subjective. Gradation from 1 plus to 4 plus turbidity was made on the basis of the appearance of the entire semen specimen.

PH. Nitrazine paper was used to determine the acid-base reaction of the semen.

Count. Samples of semen were diluted 1 to 20 in tap water. This dilution was made by the use of volumetric pipettes, using 1.0 cc. portions of semen when the size of specimen allowed, but lesser volumes where necessary. After thorough mixing, the count was made on a blood-counting chamber. Counts were made under the high power of the microscope. If the spermatozoa filled the field, 5 red-blood-cell or tertiary squares of the counting chamber were counted and the actual number determined by adding 6 ciphers to the sum. If the spermatozoa were sparse, all 25 red-blood-cell or tertiary squares were counted, the sum divided by 5 and 6 ciphers added for determining the actual count of spermatozoa per cc. The total count per ejaculate was determined by multiplying the count per cc. by the volume of the ejaculate.

Percentage motile and quality of motility. At the time of initial examination of the semen within 3 hours of collection, a drop was examined under the high power of the microscope. An estimate was made of the percentage of total sperm which were making active, purposeful, progressive movement through the liquid of the semen. The speed and aggressiveness of their activity was graded from 1 plus to 4 plus and listed under quality of motility.

Longevity. Specimens were kept in a refrigerator at 10° C. and examined at 4-hour intervals until their motility ceased. The length of time in hours from collection of the semen until cessation of all activity was recorded as the longevity.

Morphology. A smear was made of the fresh semen specimen using one drop on a clean glass slide. This was fixed by heating in a flame and was stained by methylene blue. The percentage of abnormal forms was counted in a total of 200 spermatozoa, and the abnormalities were listed as abnormalities of head, neck, or tail.

Physiological Procedures

VENOUS PRESSURE DETERMINATIONS

All venous pressure determinations were made in the morning with the subject in the fasting state and with a minimal of activity preceding the test. The subjects were allowed 8 to 10 minutes of supine rest immediately preceding the test and remained supine during the test.

The venous pressure apparatus consisted of a U-tube with one side about 5 cm. high and the other side 35 cm. The short arm was connected by a short length of intravenous rubber tubing to an adapter and a 20-gauge needle. The bottom of the U-tube was connected to a sterile saline reservoir. The whole apparatus was sterilized before using. By allowing sterile saline to flow into the U-tube the system was filled. The reservoir tube was then clamped and the extra saline allowed to escape through the needle until the saline level in the two sides of the U-tube was equal (zero point). The rubber tube leading to the needle was clamped and the needle was inserted into one of the antecubital veins. The clamp was removed from the tube and, without stasis, the needle was left in the vein until the saline in the long arm of the U-tube ceased to rise. The height of the saline column over the zero point was recorded. The needle was removed from the vein and from the adapter. The short arm and tubing were flushed free of blood by saline from the reservoir. A new sterile needle was put

in place and the saline levels were again adjusted to zero; the apparatus was then ready for use on the next subject.

In all cases the zero point was adjusted to 10 cm. above the litter upon which the subjects rested. This adjustment was made so that the zero point on the apparatus was approximately at the heart level. During each of the major testing periods venous pressure determinations were made on 2 occasions, 2 or 3 days apart.

AEROBIC WORK TESTS

The standard aerobic work test consisted of a 30-minute walk on a motor-driven treadmill at 3.5 miles per hour and a 10 per cent grade, with a room temperature of 78° F. and 40–50 per cent relative humidity. During the tests the subjects wore shoes and woolen socks and track shorts. All work tests were made in the morning with the subjects in the fasting state.

Work heart rates were counted for 15 seconds at 25, 28, and 30 minutes of each work period, using a stop watch and a stethoscope with the diaphragm placed over the base of the heart. The average of the three values was used for the work pulse rate for each work period. The recovery pulse rates were counted for 15 seconds at 1, 2, and 3 minutes of standing rest after the completion of the 30-minute walk.

By means of a two-way valve attached to a headgear and fitted with a standard rubber mouthpiece and connected to a calibrated 500-liter Tissot, expired air was collected during the twenty-third to twenty-eighth minutes of the 30-minute walk. The volume and temperature of the expired air were recorded, and samples were taken for analysis for oxygen and carbon dioxide content by the Haldane method. All values were corrected to standard temperature and pressure.

On some occasions 5 cc. of blood were taken from the antecubital vein for aerobic blood sugar determinations during the twenty-ninth minute of the 30-minute work period. The blood was always obtained while the subject was walking; this could be done easily by having the subject grasp a vertical pipe placed alongside the treadmill and straighten his elbow. The observer grasped the elbow from underneath with one hand to minimize the movement of the arm and inserted the needle on the syringe with the other hand. With a little practice it is possible to get venous blood samples even with a subject running on the treadmill.

Oxygen consumption during aerobic work was calculated in cc. of oxygen used per minute of work. Ventilation was expressed in liters of air expired per minute of work. Respiratory efficiency was calculated as cc. of oxygen removed per liter of ventilation.

MAXIMAL WORK TEST (HARVARD FITNESS TEST)

The maximal work test (Harvard Fitness Test) as used during the Minnesota Experiment was that described in detail by Johnson, Brouha, and Darling (1942). It consisted of running on a motor-driven treadmill at 7 miles per hour and an 8.6 per cent grade until exhaustion, or a maximum of 5 minutes. At the end of the run the subject was seated and the heart rate was counted for 1 to

1.5, 2 to 2.5, and 4 to 4.5 minutes of recovery. The score was calculated from the equation:

$$S = \frac{T}{2(\Sigma PR)} \times 100$$

where S = score, T = time of run in seconds, and ΣPR = the sum of the heart beats counted from 1 to 1.5, 2 to 2.5, and 4 to 4.5 minutes of recovery.

In the Minnesota Experiment the maximal work test was always preceded by a 20-minute walk at 3.5 miles per hour and a 10 per cent grade. Temperature in the test rooms was 78° F., and the relative humidity was 40 to 50 per cent of saturation.

MAXIMAL OXYGEN TRANSPORT TEST

In all cases the maximal oxygen transport test was preceded by a 20-minute walk at 3.5 miles per hour and a 10 per cent grade on a motor-driven treadmill at a room temperature of 78° F. and a 40 to 50 per cent relative humidity with the subject in the fasting state. At the end of the 20-minute walk the subject rested supine for 5 minutes; then an antecubital venous blood sample was taken with a minimum of stasis for standard rest blood lactate and pyruvate determinations.

At the end of the 5-minute rest period the subject was connected via a two-way valve to a calibrated 250-liter Tissot. He then ran for 3 minutes on a motor-driven treadmill at 7 miles per hour and a grade adjusted to produce work of such intensity as to approach closely the maximal work capacity. The tests were repeated two days apart with the subject running at two grades differing by 2.5 per cent. If the oxygen transport did not check for the two grades, a third trial was made at a higher grade.

During the first 1.5 minutes of the run the Tissot was flushed out three times with expired air. Expired air was then collected for exactly one minute between 1' 45" and 2' 45" of the run. At the end of the 3-minute run, the mask was removed and standing recovery pulse rates were taken for 15 seconds at 1, 2, and 3 minutes of recovery. The subject then assumed supine rest and antecubital venous blood was taken at 12 minutes post-exercise. In some cases when oxygen debt determinations were made, the mask was left in place and expired air was collected in separate Tissots with the subject at supine rest for 0 to 12 and 12 to 25 minutes of recovery. In these cases a third blood sample was taken at 25 minutes of recovery. Blood samples were analyzed for lactate and pyruvate concentrations by the methods of Edwards (1938) and Friedemann and Haugen (1943).

All expired air samples were analyzed by the Haldane method for oxygen and carbon dioxide; maximal oxygen transport was expressed as cc. of oxygen used per minute. Oxygen debt was calculated as oxygen utilization in excess of basal oxygen utilization during the period of recovery.

ELECTROCARDIOGRAPHIC PROCEDURE

All electrocardiograms were recorded in the basal state. The three standard leads were taken together with heart sound records during arrested respiration. Since amplitude changes were observed rather early in the experiment, an external voltage calibration was used before and after each series, in addition to

that incorporated in the machine (Sanborn) — that is, more than ordinary precaution was taken for the correct voltage recording. The speed was checked by means of a stop watch; a slight constant correction was necessary and this was applied in the calculation of intervals and heart rate. The following intervals were measured: duration of the P wave; duration of the PR interval; QRS interval; QT interval; and duration of the mechanical systole (syst.), calculated as the interval between the start of the major oscillations of the first heart sound and the beginning of the second heart sound. Both QT interval (electrical systole) and mechanical systole were averaged from five beats, usually in lead 2. The constant K was calculated both for QT (K_{QT}) and for mechanical systole duration (K_{syst}), using the formulas:

$$K_{QT} = \frac{QT}{\sqrt{RR}}$$

$$K_{syst} = \frac{syst}{\sqrt{RR}}$$

The average RR interval was calculated from ten beats; in addition, the shortest and longest RR intervals of the whole record were measured. Their differences, absolute and in percentage of the average heart rate, were used as a criterion of arrhythmia. The amplitudes of the P wave, the QRS complex, and the T wave were measured in all leads (cf. Pardee, 1943). The QRS axis and the T axis were calculated using Dieuaide's procedure. For estimation of the over-all magnitude of the QRS complex and the T wave, the sum of the amplitudes in leads 1, 2, and 3 was calculated. The symbols Σ_{QRS} and Σ_T are used to express these values.

After lead 2 was taken with arrested respiration, the effect of maximum inspiration was recorded in the same lead. Early and late effects, especially on intervals, were analyzed.

MISCELLANEOUS PHYSIOLOGICAL METHODS

Basal values for pulse rate, blood pressure, respiratory rate, ventilation volume, and oral temperature were all measured at the time of the determination of the basal metabolic rate (B.M.R.). The subjects rose from bed in the morning, urinated but did not dress, and walked into the metabolism room where they rested quietly in bed for 15 to 20 minutes before the start of the B.M.R. measurement. Blood pressure was determined by the auscultatory method with the standardized sphygmomanometer, the fourth phase being taken as the diastolic pressure. Pulse rate was estimated as the average of two 30-second counts, palpating the radial artery.

The basal metabolism room was kept at 77 to 78° F., with the humidity at 50 per cent relative saturation. Light was subdued and constant. Quiet and calm conditions were strictly maintained. The basal metabolism was estimated from two 8-minute runs of the Benedict-Roth type of metabolism machine (Sanborn make). When the two runs did not agree within 20 cc., the measurements were repeated. The B.M.R. was corrected to 760 mm. Hg. and 0° C. for the volume of oxygen consumption. Respiratory rates and ventilation volumes were meas-

ured on the B.M.R. records, taking the average for 2 separate minutes in each record.

Vital capacity was estimated after the end of the B.M.R. measurements, with the subject sitting in a straight chair and blowing into a standard balanced gasometer. The highest value obtained in 3 trials was recorded.

Special Senses

AUDITORY ACUITY

Auditory acuity was tested by the standard Maico D-5 audiometer (Manual for Audiometers, no date). The testing was done in a soundproofed room of the University of Minnesota radio station. Five cycle-frequencies were employed: 128, 512, 2048, 4096, and 8192 double-vibrations per second. Three ascending threshold readings, in decibels, were made at each frequency. At the control period acuity was determined for both ears; afterward only the ear which was better in control was tested.

VISUAL ACUITY

Visual acuity was determined in terms of the diameter of the smallest circle which could be seen at a distance of 15 ft. The black circles, with diameters ranging from 0.25 mm. to 11 mm., were located irregularly on a white background, 1.5 by 2.5 inches. The subjects had to identify the position of a circle on the test patch. Binocular presentation was used, without an artificial pupil; we were not interested in the "absolute" acuity values but in the possible changes due to the experimental regimen.

The test was carried out under two conditions of illumination, one with 1 foot-candle and another with 100 foot-candles on the test patch.

FLICKER FUSION FREQUENCY

A 2.5 volt flashlight bulb was used as a light source. The light was diffused through an opal glass 2.5 cm. in diameter. A rotating disc, 15 cm. in diameter, with 2 open sectors of 22.5° located opposite each other, served to interrupt the light. The test patch was viewed with both eyes through an enclosed tube 40 cm. in length.

At the start of each testing the subject was exposed to the light for ½ minute, with the flicker rate well above the discrimination level, in order to adapt to the illumination intensity of the test patch. Then the rate was reduced to a coarse flicker and increased above the fusion level; this served to refresh the subject's concept of flicker and of fusion. In three successive testing trials the flicker rate was reduced to 10 flashes per second and then increased until the fusion level was reached. The rate of increase in the flicker frequency was maintained at about 2 flickers per second and was fairly constant over the critical range. The subjects reported verbally the reaching of the fusion level.

PERCEPTUAL FLUCTUATIONS

The subjects were presented with a geometrical cube design with a reversible perspective. Each subject had his own copy of the design at each testing session. Upon the "Go" signal, he fixated on the center point and began making a tally

on a sheet of paper for each perceptual fluctuation (i.e., each time the figure reversed itself or seemed to change perceptually in any manner). In order to control blinking, at the end of each 10 seconds the word "blink" was called, at which time all the subjects would blink their eyes. At the end of 60 seconds, the "Stop" signal was given. Then each subject counted the number of fluctuations he had tallied, and prepared himself for the next trial. Three trials were given during each testing session. With adequate training the recording of the reversals did not interfere with the perceptual processes.

Voluntary Motor Performance

For the purpose of analytical laboratory investigations, voluntary motor performance can be studied in three aspects: strength, speed, and coordination. A battery of psychomotor tests similar to and in part identical with those used in this study has been described by Brožek *et al.* (1946), and they have also discussed the conditions under which the test scores are valid criteria of performance capacity.

In the period of semi-starvation each man served as his own control. For this reason it was essential to train the men to a performance plateau before the start of the experiment. Considerable time and effort were devoted to this task during the 3 months of the control period. The training data were reported, in part, by Franklin and Brožek (1947). During the experimental period the men were tested at intervals of about 3 weeks; it has been our experience that without an intervening stress a plateau performance can be maintained for months at this rate of retesting.

STRENGTH

Strength was measured by standard handgrip and back-lift dynamometers. The score for a given testing session was the average of three successive determinations, in kg. When performing the back-lift test the subject stood with unbent legs and arms and attempted to lift as much as possible by straightening his back. The length of the chain between the horizontal bar held by the subject and the spring-coil of the dynamometer was adjusted to fit the height of each individual subject. The successive lifts made during a testing session were separated by at least $\frac{1}{2}$ minute, or longer if the subject felt the need for additional rest. Similarly, an adequate rest period was provided between the three determinations of the grip strength. The individual subjects were always alone, except for the recorder, in the testing room and were free to grunt and strain without embarrassment.

SPEED

(1) *Tapping.* A paper-and-pencil modification of the two-plate tapping test, suitable for group administration, was used. The subject was required to tap with a soft, blunt pencil on a sheet of paper, alternating between the right and the left column separated by a "barrier" $1\frac{1}{2}$ inches wide. An automatic timer indicated by a loud buzz the "Get Ready," "Go," and "Stop" signals. The score is the number of taps in 10 seconds. This test was repeated three times at each testing session, with an interval of 3 to 4 minutes between successive tests.

(2) *Ball-pipe test.* The performance consisted of passing a ball bearing through a vertically held conduit pipe, 1 ft. in length, catching the ball at the bottom, and replacing it at the top (Brožek, 1944). The score is the number of times the ball bearing is passed through the pipe in 1 minute. Using individual testing units, it was possible to test a group of 6 men at a time.

(3) *Gross body reaction time.* The subject, while walking on a treadmill at a speed of 3.5 m.p.h., had to watch for a light signal appearing ahead of him and then react by bending down and striking the appropriate key. A green light was the signal for depressing the key mounted on the left side of the treadmill, a red light for depressing the right key, and a white light for depressing both keys simultaneously. Large paddle-keys were used to minimize the element of hand coordination. The score (in 1/100 sec.) is the average time per reaction. Three sets of reactions, with 25 reactions in each set, were used.

(4) *Speed of leg movement.* The subject was standing, leaning back against slightly tilted boards, with weight of the body resting on the left foot. The performance involved moving the right leg, as rapidly as possible, from the resting position forward and up until it touched a horizontal steel rod located in front of the body, 8 inches below the level of the subject's trochanter. The time from the moment the foot left the ground (the starting plate) until the leg touched the rod and broke the circuit was measured by means of a condenser discharging into a ballistic galvanometer.

The task was performed both without and with a load of 10 lbs. The load was provided in the form of 2 flat pieces of lead, attached by a leather belt to the sides of the leg at ankle height. The score is the average time of the three fastest "kicks" out of a series of ten.

COORDINATION

In the pattern-tracing test the subject traced a narrow, winding path with a stylus. The pattern was attached to a board having an incline so that the subject while walking on the treadmill could comfortably hold the stylus at a right angle to the board. The time allowed for tracing the pattern was about 45 seconds and was kept relatively constant from subject to subject and from one testing session to another to eliminate the factor of speed. When the stylus touched the sides of the pattern, the error was recorded on a counter and a timer. Thus the test performance was scored both for the number of errors and for the duration of contacts.

Intelligence

In the study of intellective performance an attempt was made to examine the effect of semi-starvation both on the "mental level," determined by power tests (without work time limits) and speed tests (with strict time limits), and on "learning ability."

The use of standardized intellective tests has all the advantages and limitations of analytical tools of psychological research. We deal, admittedly, with abstract or intellectual intelligence (Thorndike *et al.*, 1935). With motivation provided "from the outside" and maintained at a high pitch, the performance score is a fair approximation of the true intellective capacity, it is an inadequate

basis for predicting performance in self-initiated intellectual activities. Generalizations concerning learning ability and based on our test material (Crossing out 4s) must be cautious.

ARMY GENERAL CLASSIFICATION TEST (AGCT)

The AGCT was developed in 1940 by the Army personnel specialists as a test of "general intelligence" for use at Army induction stations. It was administered to our subjects to obtain a characterization of the mental level of our sample against the background of the "general population."

Form AGCT-1a was used. The test contained three types of items: vocabulary, arithmetic, and block counting, measuring the verbal, numerical, and spatial components of mental ability, respectively. The content of the test is arranged in such a way that the three types of items are repeated on progressively more difficult levels (Staff, Personnel Research Section, 1945).

The working time allowance (40 min.) and method of scoring of the U.S. Army (raw score = number of correct answers minus one third of incorrect answers) were used. The raw scores were converted into Army Standard Scores, designed to facilitate the interpretation of the test results, with 100 as the expected average for the inductee population and a standard deviation of 20.

THE I.E.R. INTELLIGENCE SCALE, CAVD

The CAVD scale (Institute of Educational Research, 1933) includes four sub-tests: Completions, Arithmetics, Vocabulary, and Directions. In the completions test the subject must supply words to make a statement true and sensible. Arithmetics includes problems which can be solved by reasoning and relatively simple computations. Vocabulary involves choosing the most adequate synonym for a given word. The directions part tests the ability to understand connected discourse. The items are grouped into 17 levels of difficulty, A to Q; in the present study only the tests on levels M, N, O, P, and Q were applied. These five levels are assembled into one test booklet for use with adults of high abstract intelligence. All levels contain the four types of sub-tests, with 10 items on each level. One point-credit is given for a correctly answered item; the maximal score for a level is 40 points and for the test as a whole 200 points.

There is no limit on the working time. Each subject was allowed as much time as he desired to complete as much as he could on each test level, which grew progressively more difficult from M through Q. Most of the men worked on the five levels of the test from 8 to 10 hours, about two hours at a time. The work periods were spread over a week, and the tests were taken by the subjects at their convenience.

Out of the five alternative test forms available (Thorndike *et al.*, 1935), the forms 2, 3, and 4 were used for testing at C, S12, and S24, respectively. The raw scores were converted into "altitude" scores by using a table provided by Thorndike *et al.* (1927, p. 371). The "altitude" of an individual's intelligence is defined by the test level (from A to Q) at which he can master 50 per cent of the test items (i.e., 20 out of 40). In practice, two adjacent levels at which the correct responses total nearly 40 (between 33 and 47) were used, and the respective

tabular values of the two scores were found and averaged. The altitude score which corresponds to level A, representing the difficulty at which over 999 per thousand adults can succeed, is 230; level Q, at which only about 10 per thousand adults obtain twenty or more points, is 430.

REPEATABLE TEST BATTERY (RTB)

For the purpose of repeated testing of intellectual performance capacity a battery of six short tests was assembled. Five of these were modeled after the tests included by the Thurstones in their factorial studies of intelligence (Thurstone and Thurstone, 1941): *Flags*, in which the subject has to check whether adjacent pictures of a flag are identical or mirror images of each other – a test of ability to perceive spatial relationships (No. 26, *ibid.*, p. 61); *First Letters*, involving recall of words starting with designated letters – a test of word fluency (No. 24, p. 60); *Word-Number Recall*, requiring memorization and recall of word-number combinations – a test of immediate memory (No. 59, p. 81); *Multiplication* of two-digit numbers by one-digit numbers – a test of number facility (No. 37, p. 67); and *Letter Series*, in which the subject has to supply a missing unit in a series of letters arranged according to a definite pattern – a test of inductive reasoning (No. 34, p. 65). The sixth test, *Number Checking*, involved comparison of pairs of numbers of varying length and was included as a measure of perceptual speed; the test is an adaptation of a sub-test in the Minnesota Vocational Test for Clerical Workers (Andrew and Paterson, 1933). The work time allotments, principles of constructing alternative forms of the tests in the battery, reliabilities, and relationships to other intellectual tests have been described elsewhere (Guetzkow and Brožek, 1947).

The subjects were given training on the battery before the start of the experiment, through continuing 22 practice sessions until their performance scores reached a plateau. The battery was administered at monthly intervals throughout the semi-starvation period and the first months of rehabilitation.

CROSSING OUT 4S

The test material here consisted of 7 columns of six-digit numbers, with 24 numbers in each column. The task was to cross out all the digits 4, occurring irregularly in the numbers. This test involves perceptual speed. It was administered in a series of successive trials, given at one testing session. The score improvements with repeated performance can be used as a measure of "learning ability." In the two testing sessions given toward the end of the semi-starvation period and after 3 months of rehabilitation, 18 trials were made, each one minute in duration, with a 90-second pause between the trials.

Personality

Characterization of the individual by a score has the advantage of definiteness and brevity. Also, it places the individual on a single-dimension continuum, such as extroversion-introversion, and makes possible the comparison of groups in terms of the mean scores.

The qualitative approach to the description of personality and its changes under stress by means of such techniques as interviews is well suited for case

studies. It has the merit of revealing the individual personality in its uniqueness, against the background of the individual's past experience and his present interests, activities, and social contacts. However, this approach is extremely cumbersome when one tries to move from the discursive description of an individual to the characterization of the group. It is nearly hopeless when one is concerned with the comparison of small groups as was the case in the rehabilitation period.

In the Minnesota Experiment both the qualitative and quantitative approach was used; ratings and inventories, as well as projective tests, were applied. Frequent contacts with the men — during testing, in supervising their work as technical and clerical assistants in the Laboratory, in formal meetings and informal "bull-sessions," and in serving bread in the mess hall and eating occasionally with them — provided valuable opportunities for observation.

INTERVIEWS

Scheduled interviews were carried out by three psychologists (Dr. J. Brožek, J. C. Franklin, and Dr. H. Guetzkow), each working with one third of the men. Dr. B. C. Schiele, a psychiatrist not directly connected with the Laboratory, also interviewed some of the men, particularly those who had serious difficulties in adjusting to the experimental regimen. The psychologists differed somewhat in aim and technique in conducting the interviews. The technique ranged from a more structured to a completely nonstructured pattern. The latter yielded little that could be used for a systematic description of the changes in the group of subjects, although it occasionally provided valuable data which facilitated insight into the psychodynamics of an individual's behavior.

The subjects were encouraged to talk freely and no strait-jacket scheme or system of questions was imposed upon them at any time, even though the various parts of the interview were recorded by one interviewer under separate headings. This particular interviewer aimed at getting a picture of the subject in a systematic and relatively stable frame of reference which could be used for longitudinal study of changes induced by semi-starvation and rehabilitation. When the subjects needed help in carrying on the conversation, attention was focused, by means of questions, on five basic categories: (1) eating habits and adjustment to the starvation regimen; (2) subjective symptoms and complaints; (3) social adjustment (within the subject group, in reference to the Laboratory Staff and duties, in friendships and social contacts outside the Laboratory); (4) participation in educational activities in the experimental unit and at the University; and (5) morale, including attitudes toward the experiment, the evaluation of the subject's resistance to the semi-starvation "stress," and his "time perspective."

DIARIES

All the subjects were instructed to keep diaries, but they were given a great deal of freedom with respect to the frequency of entries and the type of items recorded. In order to facilitate the subsequent use of the records, the men were encouraged to use only the right page of the bound diary books, to start a new page every week, and to make monthly summaries of the events. The diaries were considered confidential material, and the men, for the most part, felt free

to make the diary a true reflection of their feelings and behavior. The diary material supplements in an important way the data of quantitative psychology and, together with the interviews in the present study, provides a more colorful, realistic picture of the psychological effects of reduced food intake and subsequent refeeding.

MINNESOTA MULTIPHASIC PERSONALITY INVENTORY (MMPI)

This inventory is a psychometric instrument designed to measure the abnormal components of personality. Its scales were developed by contrasting the responses of patients exhibiting relatively clear-cut clinical syndromes with those of clinically normal individuals (Hathaway and McKinley, 1943; McKinley and Hathaway, 1943). The literature on the usefulness of the instrument in a variety of clinical situations has been cited by Gough (1946). The inventory had previously been proved to be useful in experimental work and to be sensitive to the stress of a nutritional deficiency involving a severe dietary reduction of the B complex vitamins (Brožek, Guetzkow, and Keys, 1946).

The inventory contains 550 items, such as "I am happy most of the time," "The future seems hopeless to me," "What others think does not bother me." The statements are printed on separate cards. The subject responds to each statement by placing the card in "True," "False," and "Cannot say" piles. The answers are evaluated in terms of empirically derived scales for such clinical syndromes as Depression and Hysteria. The statements which tended to discriminate between normals and the criterion group of patients are regarded as diagnostic items. In characterizing an individual we determine for each scale the number of these differential items and express the sums in terms of standard scores (normal average = 50; standard deviation = 10).

The technique is objective and reliable in the sense that all subjects are exposed to the same set of statements, that the evaluation of the subject's reactions is independent of the individual who administers the inventory to the subject, and that scores obtained on repeated administration under standard conditions give reasonably consistent results. It is realized that this does not demonstrate the truthfulness of the subjects, which is the first prerequisite of the validity of all self-descriptive adjustment inventories. However, there is little reason to doubt that this prerequisite has been satisfied in the Minnesota Experiment. The subjects had nothing to gain from introducing a bias into their responses and were impressed as to the importance of giving as accurate a picture as possible of their physical and mental status.

INVENTORY OF TEMPERAMENTAL TRAITS

The Minnesota Multiphasic Personality Inventory was developed in the frame of reference of clinical psychiatry, and the scales were designed as an aid to the diagnosis of relatively well-defined syndromes. The inventories of Guilford and Martin were designed to measure personality *traits*, defined on the basis of statistical factor-analysis studies of personality questionnaires. The inventory of factors STDCR (Guilford, 1940) measures with greater degree of discrimination than is usually the case the dimensions of personality in the area of introversion-

extroversion. The factors were described as follows: S = social introversion (shyness, tendency to withdraw from social contacts); T = thinking introversion (an inclination toward philosophizing and self-analysis); D = depression (including feelings of unworthiness and guilt); C = cycloid tendencies (fluctuations in mood, tendency toward flightiness); and R = rathymia (happy-go-lucky disposition, lively and impulsive temperament).

The STDCR inventory consists of 175 statements to be marked “Yes,” “?,” or “No”; it is scored by means of templates. The score on each trait is the sum of positive responses on the critical items; for factors T and R some items carry weights of 2 points. The raw scores can be transformed into centiles based upon some 400 students at the University of Nebraska.

TABLE 437
C-SCORES AND THE CORRESPONDING FREQUENCIES OF THE RAW
SCORES FOR THE INVENTORY OF FACTORS GAMIN
(Guilford and Martin, 1943).

C-Score	Raw Score
10	highest 1%
9	next 3%
8	next 7%
7	next 12%
6	next 17%
5	average 20%
4	next 17%
3	next 12%
2	next 7%
1	next 3%
0	lowest 1%

The inventory of factors GAMIN (Guilford and Martin, 1943) attempts to measure the following five traits: G = general liking for overt activity; A = ascendancy in social situations (leadership qualities), the opposite pole of this trait being submissiveness; M = masculinity of attitudes and interests, as opposed to femininity; I = lack of inferiority feelings (self-confidence); and N = lack of nervous tenseness and irritability.

The GAMIN inventory contains 270 items. The manual provides a table for conversion of the raw scores into scaled scores (C-scores). The relationship of the C-scores to the frequency of the original scores is given in Table 437. The norms are based on 500 college men and women. High scores on the scales G, A, I, and N are interpreted as favorable characteristics; a high score on M indicates masculinity, a low score femininity, of interests and attitudes.

SELF-RATINGS

In order to systematize the recording of complaints and to obtain an estimate of their intensity, a set of items was prepared which served as the basis for the self-ratings of the subjects on specific points, such as hunger or irritability. In their ratings the subjects used letter symbols. “N” designated a “normal” amount

of the particular characteristic, or "No," meaning its absence; "N" had a numerical value of zero. Some characteristics, such as apathy, increased during starvation; others, such as ambition, decreased. The scale for the first type of items extended from "M" (more) to "EM" (extremely more), for the other type from "L" (less) to "EL" (extremely less); the numerical values for the 2 types of items extended from 0 to +5 and from 0 to -5, respectively.

The self-ratings are open to criticism on a number of counts, one of them being that the meaning of an item varies from person to person and is subject also to change for the same individual rating himself over a period of time. For this reason the specific meaning of each item was thoroughly discussed during the standardization period, and, in addition, mimeographed sheets were provided defining the characteristics to be rated. These sheets were available to each subject every time he was making the ratings. It may be useful to include the definitions of the items included in the self-rating inventory.

Hunger pain: dull, aching, gnawing sensation in the region of the stomach.

Appetite: longing, craving, or desire for food, not referred to any special region of the body.

Palatability of diet: acceptability, savoriness, agreeability of the diet to your taste. "N" (normal) refers to "standardization" diet.

Nausea: sick to your stomach, with impulse to vomit.

Dizziness: a feeling of unstable posture and unsteadiness, possibly with "swimming" sensations in the head.

Fainting: fading of consciousness and partial or total blackout.

Tiredness: general sleepiness and fatigue, the feeling of being "poohed out."

Muscle soreness: painful muscles (without cramps).

Muscle cramps: painful, involuntary contraction of a muscle or a group of muscles.

Depressed mood: feeling "low" or "blue"; discouragement; a feeling of hopelessness and inadequacy or unworthiness.

Moodiness: fluctuations in mood; rate and extent of change from dejection to elation without apparent cause.

Irritability: "getting mad" at persons and things without much provocation; the actions and events which formerly did not annoy now do. This may be entirely subjective and not exhibited in any way in actions or speech.

Apprehension: disquieting anxiety and fear, giving a sense of uneasiness.

Apathy: a not-caring attitude; general indifference and emotional unconcern.

Ambition: amount of self-initiated purposive activity; activities started by yourself, relevant to your goals.

Self-discipline: ability to carry through your educational, experimental, and free-time undertakings.

Mental alertness: wide-awake; sensitive; reacting to what is happening around you, both in your physical and social environment. (Differs from ability to concentrate in that it is concerned with the broadness and acuteness of the person's awareness.)

Concentration: ability to focus attention on a definite task and sustain it over considerable periods of time.

Comprehension: easy understanding of discussion and reading material; evidenced in the ability to put situations together and see relationships.

COMPLAINT QUESTIONNAIRE

The self-ratings involved a judgment about the intensity of the item rated. It was felt, on both theoretical and practical grounds, that this type of information might be usefully supplemented by a questionnaire in which the subject would mark a symptom as either present or absent. The questionnaire covered essentially the same areas as the self-ratings and contained items bearing on various aspects of the physical and psychological status of the respondent; some typical questions are, "Do you have many headaches?" "Are you able to work as well as ever?" "Do you find that you do not particularly desire to mix socially with people?"

As mentioned above, the questionnaire had provision only for "Yes" or "No" answers. In some items the "Yes" response, in others "No," was considered "undesirable"; an individual's score is the number of these "undesirable" responses. The average of the scores for individuals gives an indication of the change in the group as a whole. In addition to these average scores (average number of undesirable responses per subject), the frequencies of the undesirable responses to a given item represent a useful index of the changes, with respect to that item, in the group.

MAN-BY-MAN RATINGS

As an integral part of the system of ratings, each man's deviation from the pre-starvation "normal" was evaluated by means of man-by-man ratings. The subject's status was rated on a scale from 0 (normal) to 5 (extremely deteriorated). The number of men by whom the individual subjects were rated varied from 4 to 19. However, the same group of raters was maintained for each subject throughout the experiment.

DRIVE RATINGS

In investigating the dynamics of human behavior, particularly in the starving man, three "drives" appeared most important: the drive to satisfy the need for food, the drive to release tensions related to the sexual function and to obtain sexual satisfaction, and the desire for physical activity.

The men were asked to rate the intensity of these drives on a scale from "normal" (0) to "extremely more" (+5) and "extremely less" (−5) than normal. The ratings were done weekly, using a graphical method of recording. The mid-line represented "normal" (0) and the extremes corresponded to the values of +5 and −5, respectively. An increase in the intensity of a drive was represented by a rise in the graph level, a decrease by a fall in the graph line.

In order to facilitate drive ratings, some of the behavioral manifestations of the "drives" were summarized in the form of questions to be considered by the subject when making his ratings: *Food*: To what extent does the subject of food enter into your thinking, reading, and conversation? Is food and eating important to you? Do you think about the good old days when you could get as much food as you wanted? Do you often think about your old favorite dishes? To what ex-

tent are you conscious of your need for food? *Sex*: To what an extent are you occupied with sexual thoughts and activities? How often do you date? Do you desire physical contact? How often do you masturbate? How sexual is the content of your night and day dreams? *Activity*: To what extent are you physically active and interested in doing things? Or do you tend to sit and let the world go by? Do you get satisfaction out of pure physical activity or would you rather be quiet all the time?"

Statistics

(With the assistance of Dr. H. W. Alexander.)

The data obtained in the Minnesota Experiment were treated by generally accepted methods of statistical description and evaluation. Analysis of variance was used extensively. In keeping with the general plan for a full documentation of the Minnesota Experiment in this book, it seemed desirable to describe the statistical procedures in some detail. This provides the reader with tools which may be applied to the raw data given in Appendix II but not analyzed in full in the text. The methods are not novel but until now they have been infrequently applied in experimental human biology. The following presentation, then, may be useful apart from explaining the specific analysis used in the Minnesota Experiment.

SIGNIFICANCE OF THE MEAN CHANGE BETWEEN MEASUREMENTS ON TWO OCCASIONS

The difference between the values (scores) obtained for the same individual in two testing periods may be designated as d . The statistical significance of the mean difference between the two periods is tested by the F ratio:

$$(1) \quad F = \frac{\frac{(\sum d)^2}{n}}{\frac{\sum d^2 - (\sum d)^2/n}{n - 1}}$$

where n refers to the number of subjects.

For computational purposes, Formula (1) may be written as:

$$(1a) \quad F = \frac{(n - 1) (\sum d)^2}{n \sum d^2 - (\sum d)^2}$$

This F value is associated with 1 and $(n - 1)$ degrees of freedom. It is equivalent, numerically, to the square of the t -test for the significance of mean differences between paired variates (Brožek and Alexander, 1950).

Throughout this book the F values significant at the 5 per cent level are marked with one asterisk, at the 1 per cent level with two asterisks.

THE FACTORIAL DESIGN OF THE REHABILITATION PERIOD

Factorial experiments are designed for a simultaneous study of the influence of a number of experimental factors; the scope and the inductive value of an experiment increases with the number of factors involved (Goulden, 1939, p. 151). The factorial design of the first part of the rehabilitation period (R1 to

R12) was presented in Chapter 4. With 2 factors (protein and vitamins) at 2 levels and the third factor (calories) at 4 levels, we have a total of 16 dietary combinations or "diets." Two men were placed on each of these 16 diets.

REPLICATE VARIANCE

Differences in the recovery of the paired (replicate) subjects receiving the *same* diet provide us with a measure of the "experimental error." The smaller these differences, the greater the confidence we may put in the differences between the recovery of groups receiving *different* diets. The amount of recovery was determined as the difference (Δ) between test values obtained for each of the 32 subjects in the rehabilitation period (R6, R12) and at the end of semi-starvation (S24), $\Delta = R - S24$. The variance for one pair of these Δ scores, Δ' and Δ'' , is calculated as:

$$(2) \quad V = \frac{(\Delta' - \Delta'')^2}{2}$$

It should be noted that the two recovery scores, Δ' and Δ'' , obtained for each diet-pair are interchangeable.

To obtain the replicate ("error") variance for a group or groups of subjects, with p as the total number of replicate pairs, we summate and average the variances of the individual pairs:

$$(3) \quad V_{\text{rep}} = \frac{\frac{\sum (\Delta' - \Delta'')^2}{2}}{p}$$

For computational purposes Formula (3) may be written:

$$(3a) \quad V_{\text{rep}} = \frac{\frac{n}{2} \Delta^2 - \frac{1}{2} \sum D^2}{p}$$

where n = number of individuals; p = number of pairs; Δ = an individual recovery score; $D = \Delta' + \Delta''$, the sum of the recovery scores for a pair of subjects.

The replicate variance for a caloric group containing 4 diet-pairs is obtained as:

$$(4) \quad V_{\text{rep}} = \frac{\frac{8}{2} \Delta^2 - \frac{1}{2} \sum D^2}{4}$$

The replicate variance based on all 16 diet-pairs is obtained as:

$$(5) \quad V_{\text{rep}} = \frac{\frac{32}{2} \Delta^2 - \frac{1}{2} \sum D^2}{16}$$

This value is used as the denominator in all F-tests of significance of the differences between means based on all 32 subjects, such as the means of the 16 basal-protein and 16 extra-protein subjects.

HOMOGENEITY OF THE REPLICATE VARIANCES

In comparing any 2 caloric groups, with 4 diet-pairs in each group, the average replicate variance based on 8 diet-pairs should be used as the "error" term (the denominator) in the F-test. However, if the average replicate variances for the 4 caloric groups were homogeneous, we could use the average replicate variance for the whole sample in evaluating the difference between any 2 caloric groups. By this procedure the degrees of freedom are increased (16 vs. 8) and the amount of computational work is markedly reduced.

The homogeneity of the replicate variances of the 4 caloric groups has been tested by the Chi-square test (Snedecor, 1946, p. 249), and the values of Chi-square, associated with 3 degrees of freedom, are given for a set of physiological, biochemical, and psychological characteristics in Table 438. In no case did the significance of "heterogeneity" reach the 5 per cent level, although occasionally the Chi-square came close to the critical point.

The test of homogeneity was applied above to the replicate variances ob-

TABLE 438

TEST FOR HOMOGENEITY OF REPLICATE VARIANCES OF THE 4 CALORIC GROUPS AT 12 WEEKS OF REHABILITATION (Minnesota Experiment).

Function	Chi-square
Johnson test	
Time	3.359
Score	2.064
Kgm. work/kg. weight	3.195
Hemoglobin (gm.)	0.247
Basal pulse (beats/min.)	6.452
Basal O ₂ consumption (cc./min.)	0.799
Basal diastolic blood pressure	7.076
Basal pulse pressure	4.627
Aerobic O ₂ consumption (cc./min.)	0.658 (at R6)
Aerobic pulse increment	0.527 (at R9)
Aerobic work pulse	0.319 (at R9)
Calculated body fat (kg.)	0.712
Hand dynamometer (kg.)	0.920
Reaction time, gross body	0.183
Pattern tracing	
Number of contacts	1.158
Length of contacts	1.013
Group ball-pipe test	3.140
Minnesota Multiphasic Personality Inventory	
Hypochondriasis	1.985
Hysteria	4.709
Depression	5.587
The value of Chi-square for 3 degrees of freedom	
At the 5 per cent level of significance	7.815
At the 1 per cent level of significance	11.341

tained for each of the 4 caloric groups. These groups tended to differ in the rate of recovery, and it could be expected that the magnitude of the experimental "error" would be related to the caloric level. For examining the more general question of the over-all homogeneity of the variances, we may apply the Chi-square test to the replicate variances computed for each of the 16 pairs of subjects.

SIGNIFICANCE OF THE DIFFERENCES IN THE RECOVERY OF TWO GROUPS

Significance of the differences in the recovery of 2 or more groups of subjects maintained on different caloric, protein, or vitamin levels of refeeding may be tested by an F-test, obtained as the ratio:

$$(6) \quad F = \frac{V_{\text{bGr}}}{V_{\text{rep}}}$$

where V_{bGr} is variance "between groups" and V_{rep} is the replicate (error) variance.

For 2 groups, the formula for between-group variance is:

$$(7) \quad V_{\text{bGr}} = \frac{(S_1 - S_2)^2}{2n}$$

The S values refer to the sums of the recovery scores, Δ , for the 2 groups being compared; n is the number of subjects in each group ($n_1 = n_2 = n$).

SIGNIFICANCE OF THE DIFFERENCES BETWEEN THE FOUR CALORIC GROUPS

The effect of caloric levels may be tested by comparing 2 caloric groups at a time. The procedure was described above. In testing *simultaneously* the significance of the differences between all 4 caloric groups, the between-group variance is obtained according to the formula:

$$(8) \quad V_{\text{bGr}} = \frac{\frac{\sum_{k=1}^k (S^2)}{n} - \frac{(GS)^2}{nk}}{k - 1}$$

where S = the sum of Δ 's for a group; $GS = \sum_{k=1}^k S$ = the grand sum; n = the number of subjects in a group; and k = the number of groups. The between-group variance is compared again with the replicate (error) variance, as indicated in Formula (6).

The ordinary F-test takes into account the magnitude but not the order (direction) of mean differences. Only inspection of the means can tell us whether or not the amount of recovery parallels the caloric intake. The F-test indicates simply whether the differences between the means are significantly larger than could be expected "by chance" — that is, in comparison with the amount of fluctuation in the recovery scores of pairs of subjects receiving *identical* diets.

INTERACTION BETWEEN DIETARY TREATMENTS

As indicated above, 3 treatment factors were present in the rehabilitation phase of the Minnesota Experiment: calories (at 4 levels – Z, L, G, and T), proteins (at 2 levels – supplemented, Y, and unsupplemented, U), and vitamins (also at 2 levels – supplemented, H, and unsupplemented, P). At each caloric level there were 4 dietary combinations: UP, UH, YP, and YH.

It is conceivable that each dietary "treatment" might be more effective in combination with other treatment or treatments. This is referred to as "interaction" between treatments. The interaction variances (mean squares) are obtained, like any other variance, by dividing the sum of squared deviations by the degrees of freedom (df). The sums of squared deviations will be indicated by the symbol ss.

In dealing with 2 treatment factors, each factor being present at 2 levels, one compares the recovery scores for subjects who received neither or both treatments and the recovery scores for subjects who received one but not the other treatment. For proteins and vitamins we have:

$$(9) \quad SS_{\text{Prot.} \times \text{Vit.}} = \frac{[(UP + YH) - (UH + YP)]^2}{4m}$$

where UP and YH, YP and UH represent the sums of recovery scores for the respective diet groups and m refers to the number of variates entering into each sum; for all caloric groups combined $m = 8$. This interaction variance is associated with one degree of freedom. Consequently, the value of $V_{\text{Prot.} \times \text{Vit.}} = SS_{\text{Prot.} \times \text{Vit.}}$

The formula for the sum of squared deviations for interaction of proteins and calories is:

$$(10) \quad SS_{\text{Prot.} \times \text{Cal.}} = \frac{\sum_{Z}^T (Y_c - U_c)^2}{2m_c} - \frac{(Y - U)^2}{2m}$$

where U_c and Y_c refer to the sums of the recovery scores of the 4 basal-protein and the 4 extra-protein subjects in each caloric group ($m_c = 4$) and U and Y refer to the total sums based on 16 subjects ($m = 16$). The variance is associated with $(k - 1)$ degrees of freedom, where $k =$ number of caloric groups:

$$(11) \quad V_{\text{Prot.} \times \text{Cal.}} = \frac{SS_{\text{Prot.} \times \text{Cal.}}}{(k - 1)}$$

Similarly, for the interaction of vitamins and calories:

$$(12) \quad SS_{\text{Vit.} \times \text{Cal.}} = \frac{\sum_{Z}^T (H_c - P_c)^2}{2m_c} - \frac{(H - P)^2}{2m}$$

$$(13) \quad V_{\text{Vit.} \times \text{Prot.}} = \frac{SS_{\text{Vit.} \times \text{Prot.}}}{(k - 1)}$$

Finally, one may compute a second order interaction, the interaction between all 3 treatment factors. The sum of the deviations squared and the variance are calculated according to Formula 14 ($m_c = 2$, $m = 8$).

$$(14) \quad SS_{\text{Prot.} \times \text{Vit.} \times \text{Cal.}} = \frac{\sum \left[\frac{\sum [(UP_c + YH_c) - (UH_c + YP_c)]^2}{4m_c} - \frac{[(UP + YH) - (UH + YP)]^2}{4m} \right]}{1}$$

$$(15) \quad V_{\text{Prot.} \times \text{Vit.} \times \text{Cal.}} = \frac{SS_{\text{Prot.} \times \text{Vit.} \times \text{Cal.}}}{(k - 1)}$$

The concept of interaction will be presented in greater detail, in connection with concrete examples, in the following discussion.

ANALYSIS OF VARIANCE IN REHABILITATION — AN EXAMPLE

The body weight increments after 12 weeks of rehabilitation obtained for the 16 dietary combinations, with 2 subjects placed on each of these diets, are given in Table 439. The table contains also various totals obtained from the individual weight increments. The values designated as x and $\sum(xS)$ will not be used in the present section.

The variance for one pair of subjects is obtained by squaring the difference between the two recovery scores and dividing by 2 (see Formula 2). The variances are indicated in Table 440.

Applying Bartlett's test of homogeneity to these 16 variances, we obtain a Chi-square value of 16.705 before correction and 12.336 after correction. The obtained Chi-square, associated with $(16 - 1)$ degrees of freedom, was lower than the value corresponding to the 5 per cent level of probability (24.996). Consequently, the variances can be considered as essentially homogeneous. The average replicate (error) variance is obtained from Table 440 as:

$$(16) \quad V_{\text{rep}} = \frac{48.30}{16} = 3.02$$

This "error" term, associated with 16 degrees of freedom, is to be used in testing the significance of differences between two or more groups by means of the F-test:

$$(17) \quad F = \frac{\text{variance "between groups" (or "interaction" variance)}}{\text{"replicate" (error) variance}}$$

The variances (mean squares) together with their degrees of freedom are indicated in Table 441.

A glance at Table 441 shows that only one variance (mean square) reaches the level of significance when compared with the replicate variance (error mean square). The mean square for calories, 44.13, divided by 3.02 yields 14.62, a

TABLE 439

WEIGHT INCREMENTS, Δ , IN KG., OF 16 PAIRS OF SUBJECTS MAINTAINED FOR 12 WEEKS ON EXPERIMENTALLY VARIED REHABILITATION DIETS; also derived sums (S), totals of the sums (ΣS), and $\Sigma(xS)$ values. (Minnesota Experiment.)

Protein	Vitamin	Weight Increments (kg.)			
		Z	L	G	T
Basal	Basal	4.2	4.6	10.3	9.5
		2.1	4.8	5.6	9.2
Basal	Extra	4.1	5.1	7.0	8.3
		2.1	6.1	6.3	10.3
Extra	Basal	5.1	5.3	8.1	11.0
		2.3	6.0	4.2	10.1
Extra	Extra	3.8	7.1	10.4	6.1
		3.8	2.9	6.1	6.1

Protein	Vitamin	Derived Sums (S)				ΣS	$\Sigma(xS)$
		Z	L	G	T		
Basal	Basal	6.3	9.4	15.9	18.7	50.3	43.7
Basal	Extra	6.2	11.2	13.3	18.6	49.3	39.3
Extra	Basal	7.4	11.3	12.3	21.1	52.1	42.1
Extra	Extra	7.6	10.0	16.5	12.2	46.3	20.3
Basal		12.5	20.6	29.2	37.3	99.6	83.0
Extra		15.0	21.3	28.8	33.3	98.4	62.4
	Basal	13.7	20.7	28.2	39.8	102.4	85.8
	Extra	13.8	21.2	29.8	30.8	95.6	59.6
Caloric sums		27.5	41.9	58.0	70.6	198.0	145.4

Values of multiplier x	-3	-1	1	3
------------------------	-------	----	----	---	---

TABLE 440

VARIANCES FOR THE 16 PAIRS OF SUBJECTS, derived from data in Table 439.

Protein	Vitamin	Caloric Group			
		Z	L	G	T
Basal	Basal	2.205	0.020	11.045	0.045
Basal	Extra	2.000	0.500	0.245	2.000
Extra	Basal	3.920	0.245	7.605	0.405
Extra	Extra	0.000	8.820	9.245	0.000

TABLE 441

ANALYSIS OF VARIANCE, WITHOUT REGRESSION.

Source of Variation	Degrees of Freedom	Sum of Squared Deviations	Variance (mean square)
Error (between replicate subjects)	16	48.30	3.02
Proteins (P)	1	.04	.04
Vitamins (V)	1	1.44	1.44
Proteins × Vitamins (PV)	1	.72	.72
Calories (C)	3	132.40	44.13
Proteins × Calories (PC)	3	2.82	.94
Vitamins × Calories (VC)	3	9.03	3.01
Proteins × Vitamins × Calories (PVC) . . .	3	15.95	5.32
Total	31	210.70	

TABLE 442

AVERAGE WEIGHT INCREMENTS DERIVED FROM TABLE 439.

Protein	Vitamin	Caloric Group				Mean
		Z	L	G	T	
Basal	Basal	3.15	4.70	7.95	9.35	6.29
Basal	Extra	3.10	5.60	6.65	9.30	6.16
Extra	Basal	3.70	5.65	6.15	10.55	6.51
Extra	Extra	3.80	5.00	8.25	6.10	5.79
Basal		3.12	5.15	7.30	9.32	6.22
Extra		3.75	5.32	7.20	8.32	6.15
	Basal	3.42	5.18	7.05	9.95	6.40
	Extra	3.45	5.30	7.45	7.70	5.98
Mean		3.44	5.24	7.25	8.82	6.19

highly significant F value, with 3 and 16 degrees of freedom; F value at the 1 per cent level of significance, $F_{0.01} = 5.29$. The implication is that the differences in caloric intake were associated with significant differences in weight increment. This is an over-all statement which requires further analysis. Methods will be presented below for the study of the nature of the interdependence between these 2 variables. The F-tests of Table 441 must be interpreted with reference to the means of the different groups compared. The mean values are given in Table 442.

It may be worth while to provide a graphic interpretation for the “interaction” components of the analysis of variance in Table 441. In Figures 146, 147, 148, and 149 the ordinate represents average weight increments. The “interaction” between proteins and vitamins is a measure of the lack of parallelism between the two lines of Figure 146; if the interaction were zero, the lines would be parallel. From Table 441 we find that the Prot. × Vit. interaction is nonsignificant, which means that the lack of parallelism in Figure 146 is attributable to random variation. Absence of interaction between proteins and vitamins

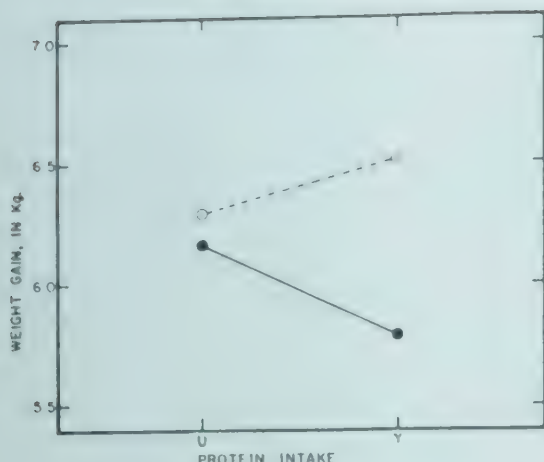


FIGURE 146. INTERACTION OF PROTEINS AND VITAMINS. Open circles indicate the weight gains of subjects who received no vitamin supplements (symbol P, "Placebo"); solid circles refer to vitamin-supplemented subjects (symbol H, "Hexavitamin"). U = unsupplemented with protein; Y = supplemented with protein ("Yes"). (Minnesota Experiment.)

FIGURE 147. INTERACTION OF PROTEINS AND CALORIES. Open circles indicate weight gains for subjects not receiving protein supplements (U); solid circles refer to protein-supplemented subjects (Y). (Minnesota Experiment.)

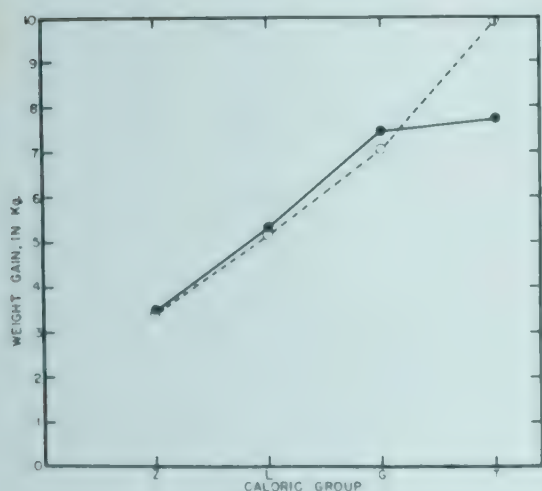
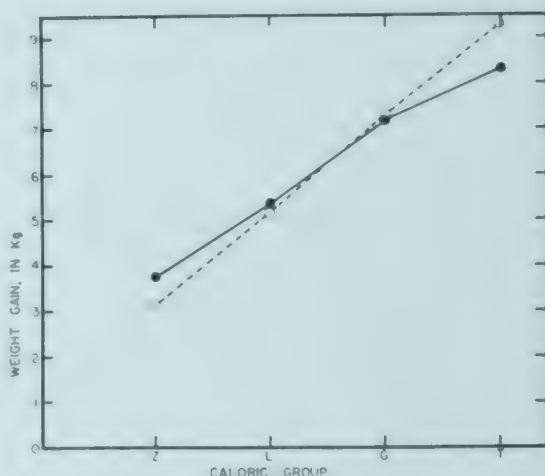
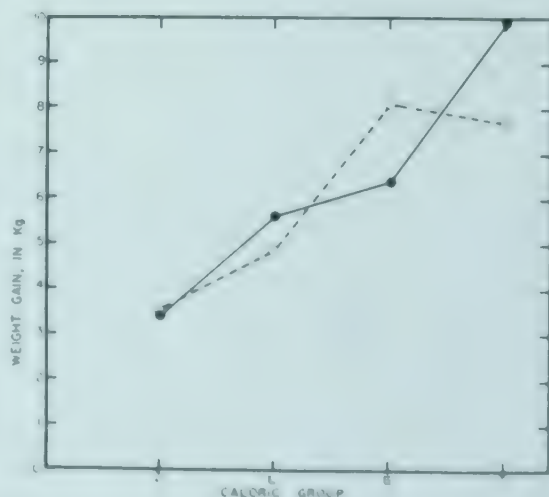


FIGURE 148. INTERACTION OF VITAMINS AND CALORIES. Open circles indicate weight gains for subjects not receiving vitamin supplements (P); solid circles refer to vitamin-supplemented subjects (H). (Minnesota Experiment.)

FIGURE 149. INTERACTION OF PROTEINS, VITAMINS, AND CALORIES. Open circles indicate average weight gains of subjects not supplemented with proteins and vitamins (UP) and of subjects supplemented with both (YH); solid circles refer to subjects supplemented with either proteins (YP) or vitamins (UH). (Minnesota Experiment.)



means that the 2 factors act additively — that is, the effect of protein supplementation *and* vitamin supplementation is equal to the sum of the effects of these 2 factors taken singly.

In Figures 147 and 148 the abscissa represents the caloric intake; the distances between caloric groups were assumed to be equal. The ordinate indicates weight increments at the 2 protein levels (Figure 147) and the 2 vitamin levels (Figure 148). The interaction Prot. \times Cal. measures the lack of parallelism between the two graphs of Figure 147, while the Vit. \times Cal. interaction measures the lack of parallelism between the two graphs of Figure 148.

Figure 149 provides a similar interpretation for Prot. \times Vit. \times Cal. interaction. The values indicated by open circles were obtained by averaging the recovery scores for subjects on low protein, low vitamin (UP) and high protein, high vitamin (YH) diets. The values indicated by solid circles were obtained similarly from the average of low protein, high vitamin (UH) and high protein, low vitamin (YP) diets. The interaction is a measure of the lack of parallelism of these two graphs, or a measure of the additivity in the 3 factors (proteins, vitamins, and calories).

REGRESSION ANALYSIS

It has been pointed out that in testing the significance of differences between the recovery of the subjects in the 4 caloric groups only the magnitude of the differences between group means was taken into account. The actual mean recovery scores for the 4 caloric groups, from Z to T, were 3.44, 5.24, 7.25, and 8.82 kg. It should be noted that the value of the variance "between caloric groups" (and the resulting F-test) would have been the same irrespective of the order of the means. Obviously, in interpreting the biological significance of the obtained differences we do consider the order of the means of the recovery scores and relate them to the caloric levels.

Statistically, the order of the means is taken into account by expressing the relationship between caloric supplements and the weight increments in terms of the regression analysis. Consider for the time being the average weight gains for the 4 caloric groups, with 8 subjects in each group. The weight gains, taken from Table 442, are plotted against the level of caloric intake in Figure 150. In addition, a least-square regression line has been fitted to the mean weight gains.

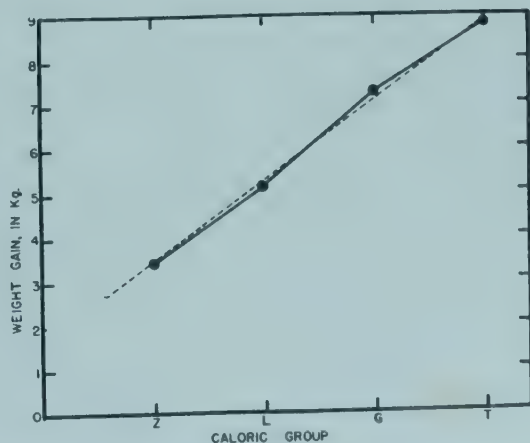


FIGURE 150. ACTUAL WEIGHT GAINS OF THE 4 CALORIC GROUPS AFTER 12 WEEKS OF REHABILITATION (solid circles) and a least-square line fitted to these means (Minnesota Experiment).

TABLE 443
LINEAR ESTIMATES DERIVED FROM TABLE 442.

Protein	Vitamin	Caloric Group				Mean
		Z	L	G	T	
Basal	Basal	3.01	5.20	7.38	9.56	6.29
Basal	Extra	3.22	5.18	7.14	9.11	6.16
Extra	Basal	3.36	5.46	7.56	9.67	6.51
Extra	Extra	4.26	5.28	6.30	7.31	5.79
Basal		3.11	5.19	7.26	9.34	6.22
Extra		3.81	5.37	6.93	8.49	6.15
	Basal	3.18	5.33	7.47	9.62	6.40
	Extra	3.74	5.23	6.72	8.21	5.98
Mean		3.46	5.28	7.10	8.91	6.19

TABLE 444
RESIDUALS DERIVED FROM TABLES 442 AND 443.

Protein	Vitamin	Caloric Group			
		Z	L	G	T
Basal	Basal	.14	-.50	.57	-.22
Basal	Extra	-.12	.42	-.50	.19
Extra	Basal	.34	.19	-1.42	.88
Extra	Extra	-.46	-.28	1.96	-1.21
Basal		.01	-.04	.04	-.01
Extra		-.06	-.04	.27	-.16
	Basal	.24	-.15	-.42	.33
	Extra	-.29	.07	.73	-.51
Mean		-.02	-.04	.15	-.09

The linear estimates for values in the rows of Table 442 are given in Table 443. The differences between the obtained and the estimated values are referred to as residual values and are indicated in Table 444.

The table of linear estimates contains the linear component of the weight increments, while the table of residuals contains what is left over after the linear component has been removed. If the data would warrant such a procedure, quadratic and cubic components could also be determined. In the case of weight increments, and in the majority of other functions studied during the rehabilitation period as well, the linear term accounts for a very large part of the recovery values. In the following analysis only the linear component and the residual (deviation from linear) component will be considered.

Applying the analysis of variance to these weight gain components, we separate the over-all effect of calories into the *linear* (L) effect and the *residual* (R) effect. Consequently, each of the 4 terms in Table 441 involving calories (Cal., Prot. \times Cal., Vit. \times Cal., and Prot. \times Vit. \times Cal.) will give rise to 2 terms: L and R, Prot. \times L and Prot. \times R, Vit. \times L and Vit. \times R, Prot. \times Vit.

× L and Prot. × Vit. × R. The comparison of the weight gains of the basal-protein and extra-protein groups and of the basal-vitamin and extra-vitamin groups is not affected.

It is readily possible to construct graphs, similar to those given in Figures 146 to 149 but based on the linear and the residual components (given in Tables 443 and 444); such figures would provide a geometrical interpretation for all the new terms in the analysis. The term V_L when compared with the error (replicate) variance indicates whether the regression line obtained for the means of the 4 8-man caloric groups has a significant slope; other linear terms measure whether there is a significant difference between the slope of the respective 2 regression lines. Similarly, V_R indicates whether the mean residual values for the 4 caloric groups differ significantly from each other, and the residual interaction terms measure whether the respective 2 sets of residual values differ significantly.

COMPLETE ANALYSIS OF VARIANCE, INCLUDING REGRESSION

In actual practice it is not necessary to calculate the linear estimates and the residuals. The complete analysis may be conveniently carried out by means of a scheme based on a 13-block pattern indicated in Table 439. In addition to the sums (S) and the sum totals (ΣS), the last row of values, designated as x, and the sum of the products, $\Sigma(xS)$, will be used. The next step involves computation of the values presented in Tables 445 and 446.

The directions for combining the values of Table 446 in order to obtain the "sums of squared deviations" (ss) and the "degrees of freedom" (df), needed for computing the variances (mean squares), are given compactly in Table 447.

TABLE 445

INTERMEDIARY VALUES DERIVED FROM TABLE 439. The numerators represent the sums of the squares of items in the 13 boxes of Table 439. The denominators for the 5 blocks on the left in the present table and the 4 blocks in the middle refer to the number of items which entered into each item of Table 439. The denominators for the 4 blocks on the right represent the product of denominators in the left-hand column of blocks in the present table, multiplied by $\Sigma x^2 = 20$.

1,435.84		
1		
2,775.08	9,816.68	5,638.68
2	8	2×20
5,441.56	19,602.72	10,782.76
4	16	4×20
5,472.02	19,625.17	10,913.80
4	16	4×20
10,860.22	39,204.00	21,141.16
8	32	8×20

TABLE 446
SUMS (Σ) DERIVED FROM TABLE 445 AND THE NUMBERS (N)
OF ITEMS IN EACH BLOCK.

$\Sigma = 1435.84$		
$N = 32$		
$\Sigma = 1387.54$	$\Sigma = 1227.335$	$\Sigma = 140.97$
$N = 16$	$N = 4$	$N = 4$
$\Sigma = 1360.39$	$\Sigma = 1225.17$	$\Sigma = 134.78$
$N = 8$	$N = 2$	$N = 2$
$\Sigma = 1368.005$	$\Sigma = 1226.57$	$\Sigma = 136.42$
$N = 8$	$N = 2$	$N = 2$
$\Sigma = 1357.53$	$\Sigma = 1225.125$	$\Sigma = 132.13$
$N = 4$	$N = 1$	$N = 1$

It contains 16 "codes," each consisting of 0, +, and - signs which indicate how the values of Table 446 are to be combined. Thus to obtain the df for Prot. \times Vit., we work with the N values in the 4 blocks of the middle column of Table 446 and combine them as indicated by the signs:

$$\text{df for Prot.} \times \text{Vit.} = 4 - 2 - 2 + = 1$$

$$\text{ss for Prot.} \times \text{Vit.} = 1227.335 - 1225.17 - 1226.57 + 1225.125 = 0.72$$

The method of deriving the codes and the logic behind the codes were discussed in detail in connection with an analysis of other data (Alexander, 1946).

The application of these codes to the data of Table 446 provides all the variances (mean squares) needed for the analysis. These data are given in Table 448. This is a comprehensive table including the error term (replicate variance), the "between protein groups," "between vitamin groups," and Prot. \times Vit. interaction terms (presented earlier in Table 441), and the 8 terms resulting from the analysis of variance applied in conjunction with the regression analysis.

Comparison of the values referring to the effect of calories, alone or in combination, in Tables 441 and 448 will disclose that the sums of squares and the degrees of freedom of Table 441 have been divided in Table 448 into 2 parts, associated with the linear and the residual component of the weight gains, respectively.

Of all the 11 terms in Table 448, only the mean square for the linear effect of the calories yields a significant F-test ($F = 132.13/3.02 = 43.75$); the 1 per cent level of F for 1 and 16 degrees of freedom is 8.53. One must draw the conclusion that only the differences in caloric levels—not those in the other two factors, proteins and vitamins—significantly affected the weight gains and that the relationship between caloric intake and the weight increments, within the limits and conditions of the experiment, was linear. There was no significant interaction between the three dietary factors: calories, proteins, and vitamins.

In the present case the refined regression analysis did not alter in any way the conclusions reached on the basis of the analysis of variance without regression (Table 441), combined with inspection of the weight recovery graphs. The

TABLE 447

CODES FOR COMBINING THE SUMS (Σ) AND NUMBERS (N) OF TABLE 446. The combination of Σ ' values yields "sums of squared deviations"; the combination of N 's yields the corresponding "degrees of freedom."

E			P			V			PV		
+			0			0			0		
—	0	0	0	0	0	0	0	0	0	+	0
0	0	0	0	+	0	0	0	0	0	—	0
0	0	0	0	0	0	0	+	0	0	—	0
0	0	0	0	—	0	0	—	0	0	+	0
C			PC			VC			PVC		
0			0			0			0		
0	0	0	0	0	0	0	0	0	+	—	0
0	0	0	+	—	0	0	0	0	—	+	0
0	0	0	0	0	0	+	—	0	—	+	0
+	—	0	—	+	0	—	+	0	+	—	0
L			PL			VL			PVL		
0			0			0			0		
0	0	0	0	0	0	0	0	0	0	0	+
0	0	0	0	0	+	0	0	0	0	0	—
0	0	0	0	0	0	0	0	+	0	0	—
0	0	+	0	0	—	0	0	—	0	0	+
R			PR			VR			PVR		
0			0			0			0		
0	0	0	0	0	0	0	0	0	+	—	—
0	0	0	+	—	—	0	0	0	—	+	+
0	0	0	0	0	0	+	—	—	—	+	+
+	—	—	—	+	+	—	+	+	+	—	—

TABLE 448

COMPLETE ANALYSIS OF VARIANCE, WITH REGRESSION.

Source of Variation	Degree of Freedom	Sum of Squared Deviations	Variance (mean square)
Error (between replicate subjects) (E)	16	48.30	3.02
Proteins (P)	1	.04	.04
Vitamins (V)	1	1.44	1.44
Proteins \times Vitamins (PV)	1	.72	.72
Calories			
Linear component (L)	1	132.13	132.13
Residual component (R)	2	.27	.14
Proteins \times Linear (PL)	1	2.65	2.65
Proteins \times Residual (PR)	2	.17	.08
Vitamins \times Linear (VL)	1	4.29	4.29
Vitamins \times Residual (VR)	2	4.74	2.37
Proteins \times Vitamins \times Linear (PVL)	1	1.89	1.89
Proteins \times Vitamins \times Residual (PVR)	2	14.06	7.03
Total	31	210.70	

latter technique has been used for the analysis of the bulk of the data, with emphasis on comparison of 2 caloric groups at a time. The over-all F-tests of the significance of the differences between the 4 caloric groups and the regression analysis of the caloric effects were carried out for a group of selected functions and characteristics in Chapter 50 (Table 435).

APPENDIX II

Detailed Data from the Minnesota Experiment

Photographs



FIGURE 151. SUBJECT NOS. 26, 20, 111, AND 101 SUN-BATHING DURING THE FINAL WEEK OF SEMI-STARVATION. Photographed by *Life* photographer Wallace Kirkland. Copyright *Time, Inc.*



FIGURE 152. THREE VIEWS OF SUBJECT NO. 20 DURING THE CONTROL PERIOD.



FIGURE 153. THREE VIEWS OF SUBJECT NO. 20 DURING THE LAST WEEK OF SEMI-STARVATION.



FIGURE 154. THREE VIEWS OF SUBJECT NO. 126 DURING THE CONTROL PERIOD.

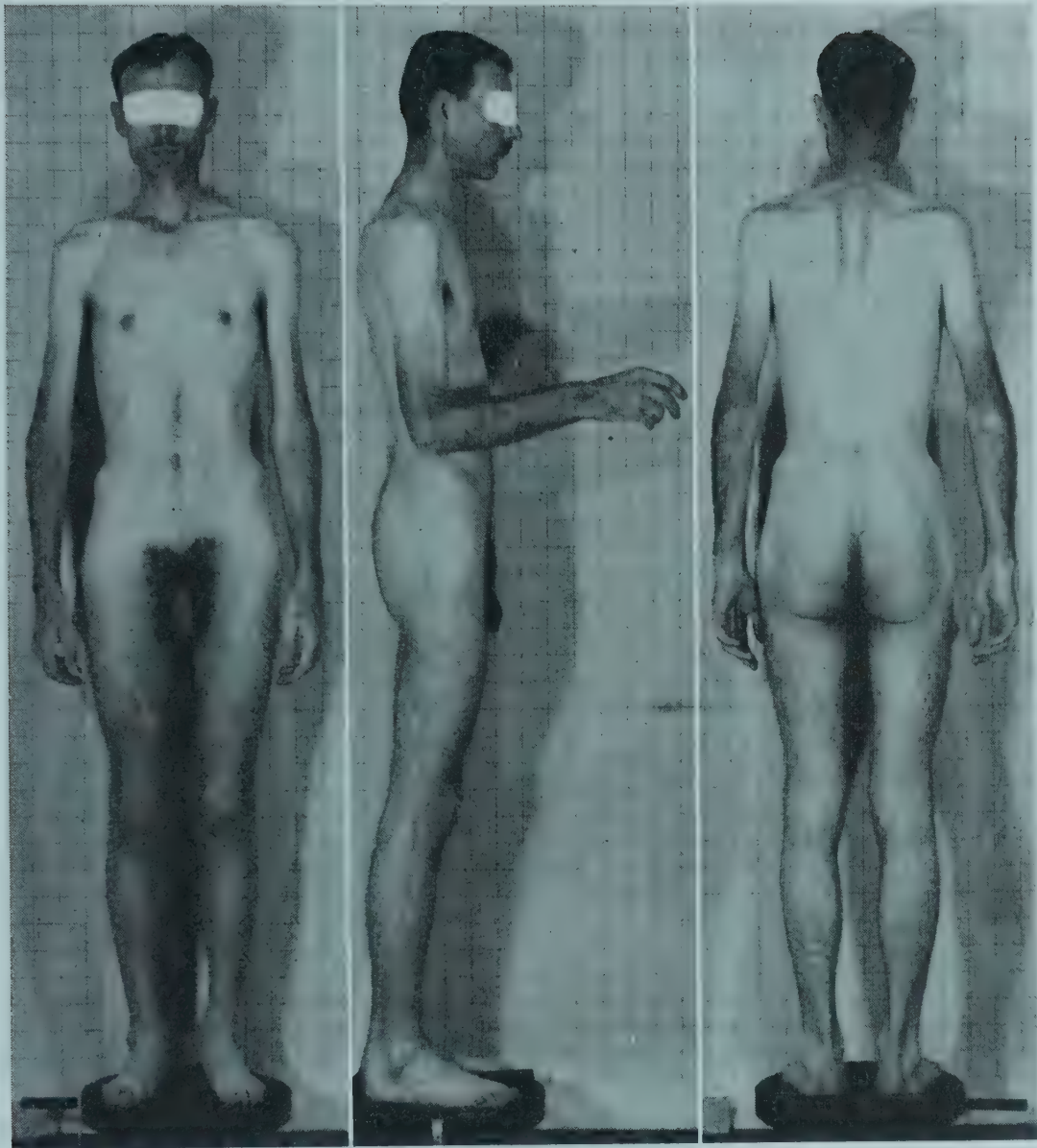


FIGURE 155. THREE VIEWS OF SUBJECT NO. 126 DURING THE LAST WEEK OF SEMI-STARVATION.



FIGURE 156. TWO VIEWS OF SUBJECT NO. 4 DURING THE CONTROL PERIOD, THE LAST WEEK OF SEMI-STARVATION, AND THE TWELFTH AND TWENTIETH WEEKS OF REHABILITATION.



FIGURE 157. FRONT AND SIDE VIEWS OF SUBJECT NO. 104 AND FRONT VIEW OF SUBJECT NO. 123 DURING THE CONTROL PERIOD, THE LAST WEEK OF SEMI-STARVATION, AND THE TWELFTH WEEK OF REHABILITATION.



FIGURE 158. FRONT VIEWS OF SUBJECT NOS. 23, 119, AND 105 DURING THE CONTROL PERIOD, THE LAST WEEK OF SEMI-STARVATION, AND THE TWELFTH WEEK OF REHABILITATION.

Tables of Measurements

THE tables of measurements provide detailed data which might be used by other investigators for further analysis. The figures are given partly as raw data and partly as differences between various stages of the experiment.

It is technically impossible to include all the material. In the selection we were guided by the principle that all available control values should be given. The semi-starvation and rehabilitation data are included only in those cases where the functions changed markedly during semi-starvation.

Methods. Reference to or descriptions of the procedures used in securing the values given in these tables are indicated either in the text or in Appendix I.

Column headings. In all the tables the following column headings indicate the phase of the Minnesota Experiment during which the observation was made: C = control period (November 18, 1944, through February 11, 1945), S = semi-starvation period (February 12, 1945, through July 29, 1945), R = rehabilitation period (beginning July 30, 1945).

The number following the letters C, S, and R indicates the approximate week of the control, semi-starvation, and rehabilitation periods when the observations were made. Because of the large number of measurements, the time required for the completion of all tests during each of the major testing periods extended over a period of days or, in some cases, weeks. If the letter C is not followed by a number, the values were secured during the last 2 to 3 weeks of the control (pre-starvation) period.

The differences (d) and (Δ) are computed as follows: $dS24$ is the S24 value minus the control (C) value; $\Delta R6$ is the R6 value minus the S24 value; and $\Delta R12$ is the R12 value minus the S24 value. In each case, the sign of the difference indicates the direction of the change during that period.

When the mean values for two periods are subtracted, the answer may not be exactly the same as that given in the column for the differences. This discrepancy is due to the rounding of the figures.

In a number of cases the testing or analytical procedure was repeated two or three times at each observation period. Under these circumstances the value used in the table for the individual subject is the mean of the separate determinations. The means routinely contain one more significant figure than appeared in the original data.

Grouping of the subjects. The subjects are grouped in the tables according to the dietary regimen used during the first 12 weeks of the rehabilitation period. There were 4 caloric groups: Z, L, G, and T. Each group was to receive about 400 Cal. more than the preceding one. The actual caloric intakes during the rehabilitation period are indicated in Table 449. Within each group half the men received extra protein and the other half received only the protein in the basal diet. Half the "extra protein" and half the "basal protein" subjects in each caloric group received extra vitamins while the others received placebos.

Statistical symbols. The means (m) were computed separately for the 4 caloric groups. In addition, the grand means (M) were computed for the entire group of 32 subjects, together with the standard deviations (SD).

TABLE 449

DAILY CALORIC INTAKE. The values are averages for each week throughout the experiment. The C Average is the mean of C10, C11, and C12; the S Average is the mean of the 24 weeks of the semi-starvation period. In the periods R1 to R6, R7 to R10, and R11 to R12, the food intake was constant; therefore, only one value is given for each period.

Supplement		Subject	Period							
Protein	Vitamin		C10	C11	C12	C Av.	S1	S2	S3	S4
CALORIC GROUP Z										
Basal	Basal	122	3187	3137	3204	3176	1658	1658	1658	1658
Basal	Basal	123	3879	3829	3896	3868	1658	1658	1658	1658
Basal	Extra	119	3416	3366	3433	3405	1658	1658	1658	1658
Basal	Extra	120	3187	3137	3204	3176	1658	1658	1658	1658
Extra	Basal	129	3879	3829	3896	3868	1658	1658	1658	1658
Extra	Basal	130	3879	3829	3896	3868	1658	1658	1658	1658
Extra	Extra	126	2953	2903	2970	2942	1658	1658	1585	1354
Extra	Extra	127	3879	3829	3896	3868	1658	1658	1658	1658
		<i>m</i>	3532	3482	3549	3521	1658.0	1658.0	1648.9	1620.0
CALORIC GROUP L										
Basal	Basal	22	3650	3600	3667	3639	1658	1658	1658	1658
Basal	Basal	23	3416	3366	3433	3405	1658	1658	1658	1658
Basal	Extra	19	2953	2903	2970	2942	1658	1658	1658	1512
Basal	Extra	20	3879	3829	3896	3868	1658	1658	1658	1658
Extra	Basal	29	3416	3249	3433	3366	1658	1658	1658	1658
Extra	Basal	30	3650	3600	3896	3715	1658	1658	1658	1658
Extra	Extra	26	3416	3366	3667	3483	1658	1658	1585	1500
Extra	Extra	27	3187	3137	3204	3176	1658	1658	1585	1354
		<i>m</i>	3446	3381	3521	3449	1658.0	1658.0	1639.8	1582.0
CALORIC GROUP G										
Basal	Basal	4	3650	3829	3896	3792	1658	1658	1658	1658
Basal	Basal	5	3073	3020	3087	3060	1658	1658	1658	1512
Basal	Extra	1	3416	3366	3433	3405	1658	1658	1658	1658
Basal	Extra	2	3299	3366	3204	3290	1658	1658	1658	1658
Extra	Basal	11	3650	3829	3896	3792	1658	1658	1658	1658
Extra	Basal	12	3879	3829	3896	3868	1658	1658	1658	1731
Extra	Extra	8	3299	3137	3204	3213	1658	1658	1585	1354
Extra	Extra	9	3879	3829	3896	3868	1658	1658	1658	1658
		<i>m</i>	3518	3526	3564	3536	1658.0	1658.0	1648.9	1610.9
CALORIC GROUP T										
Basal	Basal	104	3299	3249	3433	3327	1658	1658	1658	1658
Basal	Basal	105	3187	3137	3204	3176	1658	1658	1658	1512
Basal	Extra	101	3650	3600	3433	3561	1658	1658	1658	1585
Basal	Extra	102	3299	2903	3433	3212	1658	1658	1658	1658
Extra	Basal	111	3879	3829	3896	3868	1658	1658	1658	1658
Extra	Basal	112	3650	3829	3896	3792	1658	1658	1658	1658
Extra	Extra	108	3299	3137	3204	3213	1658	1658	1658	1658
Extra	Extra	109	3530	3480	3667	3559	1658	1658	1658	1658
		<i>m</i>	3474	3396	3521	3464	1658.0	1658.0	1658.0	1630.6
		<i>M</i>	3492.5	3446.2	3538.8	3492.5	1658.0	1658.0	1648.9	1610.9

TABLE 449 *continued*

Supplement		Subject	Period									
Protein	Vitamin		S5	S6	S7	S8	S9	S10	S11	S12	S13	S14
CALORIC GROUP Z												
Basal	Basal	122	1658	1658	1658	1658	1658	1658	1779	1828	1828	1682
Basal	Basal	123	1658	1658	1658	1658	1658	1658	1476	1330	1172	1148
Basal	Extra	119	1658	1658	1358	1658	1658	1658	1658	1658	1658	1658
Basal	Extra	120	1658	1658	1658	1658	1658	1658	1476	1476	1342	1318
Extra	Basal	129	1731	1670	1658	1731	1743	1670	1658	1512	1634	1512
Extra	Basal	130	1658	1658	1658	1658	1658	1658	1512	1658	1658	1658
Extra	Extra	126	1391	1330	1318	1245	1160	1148	1269	1318	1172	1148
Extra	Extra	127	1731	1743	1816	1828	1828	1755	1804	1682	1658	1658
		<i>m</i>	1642.9	1629.1	1635.2	1636.8	1627.6	1607.9	1579.0	1557.8	1515.2	1472.8
CALORIC GROUP L												
Basal	Basal	22	1658	1658	1658	1658	1658	1658	1537	1342	1464	1488
Basal	Basal	23	1658	1658	1658	1658	1658	1658	1658	1658	1804	1828
Basal	Extra	19	1488	1561	1500	1415	1403	1330	1318	1318	1318	1172
Basal	Extra	20	1658	1658	1658	1658	1658	1658	1658	1512	1488	1488
Extra	Basal	29	1658	1585	1427	1257	1233	1597	1901	1998	1998	1852
Extra	Basal	30	1658	1658	1658	1658	1658	1658	1537	1488	1342	1318
Extra	Extra	26	1634	1658	1658	1658	1658	1658	1658	1512	1634	1512
Extra	Extra	27	1609	1658	1658	1658	1658	1658	1658	1658	1658	1658
		<i>m</i>	1627.6	1636.8	1609.4	1577.5	1573.0	1609.4	1615.6	1560.8	1588.2	1539.5
CALORIC GROUP G												
Basal	Basal	4	1658	1658	1658	1731	1743	1743	1804	1682	1804	1682
Basal	Basal	5	1488	1561	1500	1488	1342	1318	1318	1464	1342	1318
Basal	Extra	1	1804	1828	1974	2071	2010	1852	1707	1658	1294	1148
Basal	Extra	2	1658	1658	1658	1658	1658	1658	1537	1342	1318	1464
Extra	Basal	11	1658	1658	1658	1658	1658	1658	1658	1658	1658	1658
Extra	Basal	12	1962	1998	2071	2156	2095	1937	2095	1877	1828	1682
Extra	Extra	8	1318	1245	1233	1160	1148	1148	941	858	989	1609
Extra	Extra	9	1658	1658	1658	1658	1658	1512	1427	1403	1622	1658
		<i>m</i>	1650.5	1658.0	1676.2	1697.5	1664.0	1603.2	1560.9	1492.8	1481.9	1527.4
CALORIC GROUP T												
Basal	Basal	104	1658	1585	1646	1658	1658	1512	1367	1318	1318	1172
Basal	Basal	105	1634	1585	1573	1427	1476	1342	1318	1318	1318	1318
Basal	Extra	101	1646	1658	1658	1658	1658	1658	1476	1330	1318	1318
Basal	Extra	102	1658	1658	1658	1658	1658	1658	1658	1512	1342	1464
Extra	Basal	111	1658	1658	1731	1816	1828	1755	1864	1694	1804	1682
Extra	Basal	112	1731	1675	1658	1731	1816	1755	1804	1682	1658	1512
Extra	Extra	108	1658	1585	1500	1415	1184	1148	1330	1403	1403	1330
Extra	Extra	109	1658	1658	1658	1658	1658	1658	1658	1658	1658	1658
		<i>m</i>	1662.6	1632.8	1635.2	1627.6	1617.0	1560.8	1559.4	1489.4	1477.4	1431.8
		<i>M</i>	1645.9	1639.2	1639.0	1634.8	1620.4	1595.3	1578.7	1525.2	1515.8	1492.8

TABLE 449 *continued*

Supplement		Subject	Period									
Protein	Vitamin		S15	S16	S17	S18	S19	S20	S21	S22	S23	S24
CALORIC GROUP Z												
Basal	Basal	122	1512	1342	1464	1634	1658	1658	1658	1658	1658	1658
Basal	Basal	123	1148	1148	1148	1294	1367	1488	1488	1488	1488	1488
Basal	Extra	119	1658	1367	1464	1488	1561	1573	1646	1658	1658	1658
Basal	Extra	120	1148	1342	1464	1488	1488	1488	1634	1658	1731	1743
Extra	Basal	129	1342	1318	1464	1488	1488	1488	1488	1488	1488	1488
Extra	Basal	130	1658	1658	1658	1658	1658	1221	1148	1439	1488	1634
Extra	Extra	126	1367	1148	1148	1148	945	438	584	1148	1148	1294
Extra	Extra	127	1658	1658	1512	1342	1537	1622	1658	1658	1658	1658
		<i>m</i>	1436.4	1372.6	1415.2	1442.5	1462.8	1372.0	1413.0	1524.4	1539.6	1577.6
CALORIC GROUP L												
Basal	Basal	22	1342	1172	1439	1488	1488	1488	1488	1488	1488	1488
Basal	Basal	23	1755	1974	1998	1998	1998	1852	1901	1913	1913	1913
Basal	Extra	19	1148	1148	1148	1367	1549	1646	1658	1512	1342	1464
Basal	Extra	20	1342	1172	1439	1634	1658	1658	1731	1816	1828	1974
Extra	Basal	29	1755	1974	1998	1852	1901	1986	1998	1998	1998	1998
Extra	Basal	30	1464	1318	1318	1172	1148	1148	1148	1148	1148	1148
Extra	Extra	26	1488	1318	1172	1024	858	1024	858	706	1184	1537
Extra	Extra	27	1367	1464	1488	1488	1488	1488	1488	1634	1658	1658
		<i>m</i>	1457.6	1442.5	1500.0	1502.8	1511.0	1536.2	1533.8	1526.9	1569.9	1647.5
CALORIC GROUP G												
Basal	Basal	4	1658	1804	1974	1707	1804	1828	1828	1828	1828	1828
Basal	Basal	5	1464	1318	1464	1488	1488	1488	1488	1488	1634	1658
Basal	Extra	1	1294	1609	1804	1828	1974	1949	1998	1998	1852	1925
Basal	Extra	2	1342	1172	1294	1172	1439	1549	1646	1658	1561	1634
Extra	Basal	11	1658	1512	1488	1488	1488	1561	1646	1731	1816	1828
Extra	Basal	12	1658	1512	1561	1646	1658	1658	1804	1949	2071	2083
Extra	Extra	8	1658	1512	1342	1318	1464	1634	1658	1512	1342	1464
Extra	Extra	9	1585	1342	1585	1658	1658	1658	1512	1391	1634	1658
		<i>m</i>	1539.6	1472.6	1564.0	1330.9	1621.6	1665.6	1697.5	1694.4	1717.2	1759.8
CALORIC GROUP T												
Basal	Basal	104	1148	1439	1634	1658	1658	1658	1658	1658	1658	1658
Basal	Basal	105	1464	1318	1464	1427	1622	1658	1731	1670	1658	1658
Basal	Extra	101	1172	1148	1148	1148	1148	1148	1342	1512	1792	1828
Basal	Extra	102	1634	1512	1634	1658	1658	1585	1500	1488	1488	1488
Extra	Basal	111	1658	1658	1658	1658	1658	1658	1658	1658	1658	1658
Extra	Basal	112	1488	1609	1658	1367	1318	1318	1318	815	608	1071
Extra	Extra	108	1318	1172	1148	1148	1148	1148	1148	1148	1148	1294
Extra	Extra	109	1367	1318	1464	1634	1658	1658	1731	1816	1974	1998
		<i>m</i>	1406.1	1396.8	1476.0	1462.1	1483.5	1478.9	1510.8	1470.6	1498.0	1581.6
		<i>M</i>	1459.9	1421.1	1488.8	1434.6	1519.7	1516.3	1538.8	1554.1	1581.2	1641.6

TABLE 449 *continued*

Supplement			Period				
Protein	Vitamin	Subject	S Av.	R1 to R6	R7 to R10	R11 and R12	R Av.
CALORIC GROUP Z							
Basal	Basal	122	1649.9	1977	2754	3042	2413.5
Basal	Basal	123	1468.8	1777	2554	2842	2213.5
Basal	Extra	119	1622.6	1977	2754	3042	2413.5
Basal	Extra	120	1557.3	1877	2654	2942	2313.5
Extra	Basal	129	1570.5	1984	2823	3056	2442.3
Extra	Basal	130	1595.2	1984	2823	3056	2442.3
Extra	Extra	126	1213.4	1784	2623	2856	2242.3
Extra	Extra	127	1670.7	2084	2923	3156	2542.3
		<i>m</i>	1543.6	1930.5	2738.5	2999.0	2377.9
CALORIC GROUP L							
Basal	Basal	22	1532.5	2229	3006	3294	2665.5
Basal	Basal	23	1781.0	2329	3106	3394	2765.5
Basal	Extra	19	1428.8	2129	2906	3194	2565.5
Basal	Extra	20	1624.1	2229	3006	3294	2665.5
Extra	Basal	29	1774.8	2336	3175	3408	2794.3
Extra	Basal	30	1434.4	2236	3075	3308	2694.3
Extra	Extra	26	1408.8	2236	3075	3308	2694.3
Extra	Extra	27	1583.6	2236	3075	3308	2694.3
		<i>m</i>	1571.0	2245.0	3053.0	3331.5	2692.4
CALORIC GROUP G							
Basal	Basal	4	1745.1	2696	3474	3762	3133.0
Basal	Basal	5	1483.4	2496	3274	3562	2933.0
Basal	Extra	1	1758.7	2696	3474	3762	3133.0
Basal	Extra	2	1529.5	2596	3374	3662	3033.0
Extra	Basal	11	1642.8	2704	3543	3776	3162.3
Extra	Basal	12	1833.6	2904	3743	3976	3362.3
Extra	Extra	8	1367.0	2704	3543	3776	3162.3
Extra	Extra	9	1592.7	2604	3443	3676	3062.3
		<i>m</i>	1619.1	2675.0	3483.5	3744.0	3122.6
CALORIC GROUP T							
Basal	Basal	104	1556.3	2828	3606	3894	3265.0
Basal	Basal	105	1519.4	2828	3606	3894	3265.0
Basal	Extra	101	1471.8	2928	3706	3994	3365.0
Basal	Extra	102	1591.7	2928	3706	3994	3365.0
Extra	Basal	111	1695.9	3135	3975	4208	3593.8
Extra	Basal	112	1509.3	3035	3875	4108	3493.8
Extra	Extra	108	1350.3	2835	3675	3908	3293.8
Extra	Extra	109	1659.6	3035	3875	4108	3493.8
		<i>m</i>	1544.3	2944.0	3753.0	4013.5	3391.9
		<i>M</i>	1569.5	2448.6	3257.0	3517.5	2896.2

TABLE 449 *continued*

Supplement		Subject	Period							
Protein	Vitamin		R13	R14	R15	R16	R17	R18	R19	R20
CALORIC GROUP Z										
Basal	Basal	123	4830	5836	6157	5350	5464	4800	4107	3850
Basal	Extra	119	5114	6093	4071	5280	6000	3929	3630	3800
Extra	Basal	129	4357	5621	5071	3957	4443	3800	3107	3200
Extra	Extra	127	5693	6314	5971	5400	5750	4186	3664	3571
		m	4998.5	5966.0	5317.5	4996.8	5414.2	4178.8	3627.0	3605.2
CALORIC GROUP L										
Basal	Basal	23	5371	5843	5278	6450	5421	5228	4771	4157
Extra	Extra	26	5530	6986	5085	5164	4680	3543	3607	3742
		m	5450.5	6414.5	5181.5	5807	5050.5	4385.5	4189.0	3949.5
CALORIC GROUP G										
Basal	Basal	4	5771	5657	4314	4264	3664	4036	2700	3500
Basal	Extra	2	5314	5530	5864	5343	4936	4121	•	•
		m	5542.5	5593.5	5089	4803.5	4300	4078.5	2700	3500
CALORIC GROUP T										
Basal	Basal	104	5064	5900	3971	5230	5957	4164	4493	3714
Basal	Extra	101	5079	5136	5357	4914	4243	3700	3471	3400
Extra	Basal	112	5307	4707	4186	4100	3800	3578	3200	3485
Extra	Extra	109	5200	5993	5786	7800	4950	5081	4307	4100
		m	5162.5	5434.0	4825.0	5511.0	4737.5	4130.8	3867.8	3674.8
		M	5219	5801	5092	5271	4942	4180	3732	3683

* Subject 2 was hospitalized because of influenza from R19 to R20.

TABLE 450

BODY WEIGHT, in kg., weekly averages. The body weights were obtained each morning immediately after the subject emptied his bladder. All subjects were weighed without clothing or shoes.

Supplement			Period							
Protein	Vitamin	Subject	C4	C5	C6	C7	C8	C9	C10	C11
CALORIC GROUP Z										
Basal	Basal	122	67.3	66.9	66.8	66.6	66.6	66.3	66.0	65.9
Basal	Basal	123	63.0	62.6	62.7	63.0	63.1	63.9	64.0	64.6
Basal	Extra	119	65.4	65.6	65.8	66.1	66.0	66.2	66.2	66.3
Basal	Extra	120	72.3	72.2	72.4	72.3	71.9	71.3	71.4	70.9
Extra	Basal	129	65.2	65.4	65.7	66.0	66.0	65.8	66.5	65.5
Extra	Basal	130	64.7	64.4	64.5	64.7	64.7	65.4	65.6	65.9
Extra	Extra	126	86.9	87.0	86.8	86.6	86.2	85.8	85.0	84.3
Extra	Extra	127	64.3	63.9	63.7	63.7	63.8	63.9	64.2	64.2
		<i>m</i>	68.64	68.50	68.55	68.62	68.54	68.58	68.61	68.58
CALORIC GROUP L										
Basal	Basal	22	63.5	63.5	63.3	64.0	63.8	64.1	64.6	64.9
Basal	Basal	23	70.8	70.6	70.5	69.9	69.9	69.7	69.7	69.4
Basal	Extra	19	74.5	74.1	73.2	73.2	73.3	72.8	71.9	71.5
Basal	Extra	20	63.5	63.3	63.2	63.0	63.3	63.6	64.0	64.2
Extra	Basal	29	74.0	73.7	73.3	72.9	72.8	72.4	72.1	71.4
Extra	Basal	30	67.9	67.5	67.4	67.6	68.0	67.8	68.0	68.1
Extra	Extra	26	72.3	72.2	72.0	71.8	71.4	71.6	71.3	71.3
Extra	Extra	27	77.2	76.9	77.0	76.8	76.7	76.3	76.4	75.7
		<i>m</i>	70.46	70.22	69.99	69.90	69.90	69.79	69.75	69.56
CALORIC GROUP G										
Basal	Basal	4	59.8	59.9	60.0	60.2	60.5	60.5	61.0	61.3
Basal	Basal	5	83.6	83.3	83.4	83.0	82.7	82.1	81.7	81.3
Basal	Extra	1	80.9	80.6	80.2	79.9	79.5	78.8	78.3	77.5
Basal	Extra	2	75.0	75.1	75.1	74.7	74.5	74.1	73.6	73.6
Extra	Basal	11	63.0	63.0	63.4	64.2	64.8	64.9	65.1	66.0
Extra	Basal	12	83.2	82.9	82.6	82.1	81.9	81.7	81.5	81.2
Extra	Extra	8	66.5	66.4	66.0	66.1	65.7	65.5	65.3	65.0
Extra	Extra	9	75.5	75.2	74.7	74.4	73.9	73.2	73.2	73.1
		<i>m</i>	73.44	73.30	73.18	73.08	72.94	72.60	72.46	72.38
CALORIC GROUP T										
Basal	Basal	104	70.1	69.8	70.1	69.7	69.3	69.0	68.5	68.1
Basal	Basal	105	70.2	70.0	69.8	69.4	69.4	69.3	69.1	68.8
Basal	Extra	101	62.6	62.7	62.6	62.8	63.3	63.9	64.3	64.4
Basal	Extra	102	68.7	69.0	69.1	69.0	68.8	69.0	68.6	68.1
Extra	Basal	111	62.4	62.2	62.2	62.3	62.3	62.7	62.6	62.8
Extra	Basal	112	62.3	61.9	61.8	61.4	61.4	61.6	61.5	61.4
Extra	Extra	108	69.0	68.8	68.7	68.5	68.1	67.6	67.5	67.4
Extra	Extra	109	80.5	80.2	80.4	80.4	80.5	79.8	79.7	79.3
		<i>m</i>	68.22	68.08	68.09	67.94	67.89	67.86	67.72	67.54
		<i>M</i>	70.19	70.02	69.95	69.88	69.82	69.71	69.64	69.51
		<i>SD</i>	7.22							

TABLE 450 *continued*

Supplement		Subject	Period							
Protein	Vitamin		C12	S1	S2	S3	S4	S5	S6	S7
CALORIC GROUP Z										
Basal	Basal	122	65.4	64.6	63.0	61.6	60.2	59.0	57.6	56.6
Basal	Basal	123	64.7	63.8	62.6	61.8	60.6	60.1	59.1	58.2
Basal	Extra	119	66.3	65.5	64.1	63.0	61.5	60.7	59.4	58.4
Basal	Extra	120	70.8	69.6	68.2	67.1	65.6	64.4	63.2	61.9
Extra	Basal	129	65.6	64.7	63.3	62.4	61.0	60.3	59.6	58.6
Extra	Basal	130	66.0	64.8	63.4	63.0	61.5	60.7	60.1	59.0
Extra	Extra	126	83.6	82.6	81.2	79.8	78.1	77.1	75.4	74.1
Extra	Extra	127	64.2	63.1	61.2	60.2	58.3	57.4	56.1	55.5
		<i>m</i>	68.32	67.34	65.88	64.86	63.35	62.46	61.31	60.29
CALORIC GROUP L										
Basal	Basal	22	65.1	64.2	62.8	61.4	60.2	59.2	58.1	57.2
Basal	Basal	23	69.2	68.3	66.6	65.4	64.0	62.8	61.6	60.4
Basal	Extra	19	70.6	69.6	68.3	67.6	65.9	64.6	63.7	62.6
Basal	Extra	20	64.7	63.7	62.5	61.5	60.1	59.0	58.0	57.1
Extra	Basal	29	71.1	69.7	68.1	67.3	66.5	65.5	65.4	64.5
Extra	Basal	30	68.1	67.1	65.6	64.6	63.1	62.3	61.0	60.4
Extra	Extra	26	71.3	70.3	69.3	67.7	65.8	65.0	63.5	62.3
Extra	Extra	27	75.2	74.5	73.2	72.0	69.6	68.4	67.0	65.5
		<i>m</i>	69.41	68.42	67.05	65.94	64.40	63.35	62.29	61.25
CALORIC GROUP G										
Basal	Basal	4	62.0	60.9	59.6	58.6	57.2	56.0	54.9	53.8
Basal	Basal	5	80.8	79.6	77.9	76.4	74.8	73.4	72.2	70.6
Basal	Extra	1	77.2	75.5	73.8	72.1	70.1	68.8	67.3	66.0
Basal	Extra	2	73.5	72.1	70.1	69.1	67.9	67.0	65.7	64.6
Extra	Basal	11	66.4	65.7	63.9	62.6	61.6	60.0	59.1	58.2
Extra	Basal	12	81.0	79.7	77.5	75.8	74.0	73.2	71.5	70.4
Extra	Extra	8	64.6	63.8	62.8	62.7	61.6	60.7	59.2	58.6
Extra	Extra	9	72.9	71.5	69.6	69.1	68.0	67.2	66.2	64.6
		<i>m</i>	72.30	71.10	69.40	68.30	66.90	65.79	64.51	63.35
CALORIC GROUP T										
Basal	Basal	104	68.1	66.7	64.7	63.9	63.0	62.5	61.1	60.2
Basal	Basal	105	68.7	67.4	66.0	65.4	63.7	63.0	61.7	61.3
Basal	Extra	101	64.5	63.7	62.4	61.6	60.2	59.2	58.2	57.2
Basal	Extra	102	67.9	67.0	65.5	64.6	63.4	62.2	61.0	59.9
Extra	Basal	111	63.0	62.5	60.6	59.4	58.1	57.2	56.0	54.9
Extra	Basal	112	61.7	60.6	58.9	58.0	56.3	55.9	54.8	53.4
Extra	Extra	108	66.9	66.0	64.6	63.5	62.0	61.4	60.6	59.8
Extra	Extra	109	79.3	78.3	76.3	75.0	73.4	72.5	70.9	69.6
		<i>m</i>	67.51	66.52	64.88	63.92	62.51	61.74	60.54	59.54
		<i>M</i>	69.39	68.35	66.80	65.76	64.29	63.33	62.16	61.11
		<i>SD</i>	5.85							

TABLE 450 *continued*

Supplement			Period							
Protein	Vitamin	Subject	S8	S9	S10	S11	S12	S13	S14	S15
CALORIC GROUP Z										
Basal	Basal	122	55.5	54.6	53.5	52.8	52.1	51.7	51.5	51.0
Basal	Basal	123	57.6	57.3	56.8	56.5	55.8	55.2	54.9	54.8
Basal	Extra	119	57.6	56.9	55.9	55.4	54.5	53.9	53.4	53.2
Basal	Extra	120	61.1	60.5	59.7	58.7	57.7	56.8	56.0	55.5
Extra	Basal	129	58.1	57.7	57.0	57.0	56.1	55.8	55.5	54.8
Extra	Basal	130	58.5	58.0	58.1	57.8	57.5	56.9	56.6	56.6
Extra	Extra	126	72.8	71.8	70.6	69.8	69.1	68.1	67.4	67.6
Extra	Extra	127	54.6	54.2	53.2	52.9	52.5	51.8	51.3	51.2
		<i>m</i>	59.48	58.88	58.10	57.61	56.91	56.28	55.82	55.59
CALORIC GROUP L										
Basal	Basal	22	56.8	56.2	55.4	55.0	53.8	53.4	53.0	52.4
Basal	Basal	23	59.6	58.5	57.6	56.9	55.8	55.0	54.6	53.9
Basal	Extra	19	61.7	60.8	59.4	58.6	57.5	56.8	56.1	55.7
Basal	Extra	20	56.4	55.5	54.9	54.7	53.9	53.0	52.8	52.1
Extra	Basal	29	63.0	60.3	57.4	55.1	54.5	55.2	55.5	54.1
Extra	Basal	30	59.3	59.0	58.2	58.6	57.6	57.0	56.2	56.0
Extra	Extra	26	61.7	60.6	59.7	59.3	58.1	57.7	58.0	56.7
Extra	Extra	27	64.4	63.1	61.9	61.5	60.8	60.3	60.0	58.8
		<i>m</i>	60.36	59.25	58.06	57.46	56.50	56.05	55.78	54.96
CALORIC GROUP G										
Basal	Basal	4	53.1	52.3	51.5	51.0	50.4	50.0	49.4	48.6
Basal	Basal	5	69.6	68.2	66.9	65.5	64.6	63.7	62.8	62.3
Basal	Extra	1	65.3	64.8	64.3	63.8	64.9	64.4	62.6	60.6
Basal	Extra	2	63.5	62.6	61.5	61.0	60.1	58.8	58.2	58.2
Extra	Basal	11	57.6	56.7	56.0	55.3	54.4	54.1	53.5	53.2
Extra	Basal	12	70.0	69.5	68.1	68.1	68.7	67.6	67.8	67.7
Extra	Extra	8	58.2	57.2	57.1	56.2	53.9	50.7	50.2	50.3
Extra	Extra	9	64.1	63.6	64.1	63.0	60.4	59.6	59.9	59.6
		<i>m</i>	62.68	61.86	61.19	60.49	59.68	58.61	58.05	57.56
CALORIC GROUP T										
Basal	Basal	104	59.3	58.9	58.0	57.6	56.6	55.8	54.9	53.8
Basal	Basal	105	59.9	59.3	58.1	57.5	56.5	55.7	55.0	54.7
Basal	Extra	101	56.5	55.8	55.0	54.5	53.6	53.1	53.3	53.1
Basal	Extra	102	59.0	58.0	57.5	57.2	56.3	55.5	54.5	55.2
Extra	Basal	111	54.3	53.9	53.0	52.9	52.1	51.8	51.3	50.9
Extra	Basal	112	52.5	51.9	51.4	50.8	50.5	50.4	50.7	50.2
Extra	Extra	108	59.8	60.5	60.0	59.1	57.4	56.5	55.8	55.7
Extra	Extra	109	68.6	67.8	66.9	66.3	65.4	64.8	64.5	63.7
		<i>m</i>	58.74	58.26	57.49	56.99	56.05	55.45	55.00	54.66
		<i>M</i>	60.31	59.56	58.71	58.14	57.28	56.60	56.16	55.69
		<i>SD</i>					4.85			

TABLE 450 *continued*

Supplement			Period								
Protein	Vitamin	Subject	S16	S17	S18	S19	S20	S21	S22	S23	S24
CALORIC GROUP Z											
Basal	Basal	122	50.0	49.4	49.0	48.5	48.2	47.7	47.8	47.2	47.4
Basal	Basal	123	53.9	53.4	53.0	52.0	51.9	51.5	52.1	52.2	52.1
Basal	Extra	119	52.2	51.4	51.0	50.8	50.5	50.3	50.7	49.8	49.1
Basal	Extra	120	54.7	54.0	53.4	53.0	52.3	52.1	51.3	51.2	51.6
Extra	Basal	129	53.8	53.4	53.4	53.3	53.2	53.0	52.8	52.8	52.2
Extra	Basal	130	55.4	55.7	55.7	55.7	55.6	54.2	53.9	54.5	53.6
Extra	Extra	126	66.0	65.7	65.4	65.3	63.0	62.0	62.6	61.6	60.6
Extra	Extra	127	50.8	50.4	49.6	49.2	48.7	49.2	48.7	49.2	49.3
		<i>m</i>	54.60	54.18	53.81	53.48	52.92	52.50	52.49	52.31	51.99
CALORIC GROUP L											
Basal	Basal	22	51.2	51.2	51.3	50.6	50.5	50.0	49.4	49.9	49.4
Basal	Basal	23	53.4	52.8	52.7	52.2	51.8	51.5	51.4	51.4	51.4
Basal	Extra	19	54.3	54.0	51.5	51.4	51.4	52.5	52.4	52.2	50.4
Basal	Extra	20	50.8	49.8	49.4	49.2	48.4	47.8	48.1	48.2	48.0
Extra	Basal	29	53.3	54.2	54.0	52.7	52.3	53.0	54.2	53.8	53.5
Extra	Basal	30	54.8	54.5	54.2	53.6	53.8	53.9	53.1	53.2	52.4
Extra	Extra	26	56.5	56.0	57.2	55.8	55.4	55.4	54.7	53.2	53.1
Extra	Extra	27	58.1	57.6	57.4	56.8	56.1	55.8	55.3	55.6	55.7
		<i>m</i>	54.05	53.76	53.46	52.79	52.46	52.49	52.32	52.19	51.74
CALORIC GROUP G											
Basal	Basal	4	47.9	48.3	48.3	47.5	47.3	47.1	47.1	47.3	47.4
Basal	Basal	5	60.7	60.0	59.6	58.8	58.6	58.1	57.8	57.2	57.1
Basal	Extra	1	59.2	58.8	57.7	57.3	56.6	56.5	56.6	58.2	57.0
Basal	Extra	2	57.1	56.5	56.2	54.6	55.1	55.2	57.2	57.9	55.9
Extra	Basal	11	52.5	51.8	51.9	50.8	50.3	50.0	49.5	49.9	49.6
Extra	Basal	12	66.2	65.2	65.1	64.9	63.8	62.9	61.8	61.6	63.2
Extra	Extra	8	50.5	49.9	48.8	47.8	47.7	48.8	48.9	48.3	47.5
Extra	Extra	9	58.0	57.3	57.6	57.1	57.5	57.4	57.2	56.6	58.1
		<i>m</i>	56.51	55.98	55.65	54.85	54.61	54.50	54.51	54.62	54.48
CALORIC GROUP T											
Basal	Basal	104	52.9	52.6	52.3	51.8	51.4	51.1	51.4	51.4	51.6
Basal	Basal	105	53.5	52.7	52.6	51.4	50.8	51.5	51.4	51.8	51.8
Basal	Extra	101	51.9	51.6	51.8	51.7	51.4	49.3	48.4	48.4	49.7
Basal	Extra	102	53.7	53.4	53.6	53.0	52.8	53.0	51.9	51.8	51.9
Extra	Basal	111	50.4	50.1	50.1	49.8	49.5	49.4	49.1	49.0	49.1
Extra	Basal	112	48.9	49.0	50.3	50.6	50.9	50.9	50.4	49.0	49.0
Extra	Extra	108	55.5	55.0	55.1	54.4	54.6	55.1	56.6	57.1	54.1
Extra	Extra	109	62.2	61.4	61.5	60.8	60.2	59.6	58.9	59.2	59.5
		<i>m</i>	53.62	53.22	53.41	52.94	52.70	52.49	52.26	52.21	52.09
		<i>M</i>	54.70	54.28	54.08	53.51	53.18	52.99	52.90	52.83	52.57
		<i>SD</i>									4.04

TABLE 450 *continued*

Supplement			Period							
Protein	Vitamin	Subject	C12	S24	R1	R2	R3	R4	R5	R6
CALORIC GROUP Z										
Basal	Basal	122	65.4	47.4	47.5	47.3	47.2	47.4	47.4	47.6
Basal	Basal	123	64.7	52.1	52.0	52.3	52.3	52.8	52.3	51.8
Basal	Extra	119	66.3	49.1	49.5	49.0	49.0	49.2	49.2	49.3
Basal	Extra	120	70.8	51.6	51.4	50.6	50.2	50.3	50.6	50.5
Extra	Basal	129	65.6	52.2	52.2	52.3	52.2	51.8	52.5	52.6
Extra	Basal	130	66.0	53.6	54.8	54.0	52.8	53.1	53.3	53.0
Extra	Extra	126	83.6	60.6	63.2	63.2	63.4	62.8	63.4	62.1
Extra	Extra	127	64.2	49.3	50.1	49.8	49.6	49.5	49.4	49.4
		<i>m</i>	68.32	51.99	52.59	52.31	52.09	52.11	52.26	52.04
CALORIC GROUP L										
Basal	Basal	22	65.1	49.4	49.6	49.9	50.1	50.4	51.0	51.0
Basal	Basal	23	69.2	51.4	51.2	50.4	50.4	50.8	51.2	51.6
Basal	Extra	19	70.6	50.4	50.0	50.1	50.5	51.4	51.7	52.2
Basal	Extra	20	64.7	48.0	49.8	49.1	49.2	48.1	50.6	50.7
Extra	Basal	29	71.1	53.5	53.8	54.6	55.3	55.7	55.4	56.1
Extra	Basal	30	68.1	52.4	53.3	54.3	54.3	54.5	54.7	54.6
Extra	Extra	26	71.3	53.1	54.5	54.9	54.5	55.1	55.2	55.2
Extra	Extra	27	75.2	55.7	56.2	55.3	54.8	54.7	55.0	55.4
		<i>m</i>	69.41	51.74	52.30	52.32	52.39	52.59	53.10	53.35
CALORIC GROUP G										
Basal	Basal	4	62.0	47.4	47.8	48.0	48.7	49.3	49.6	50.2
Basal	Basal	5	80.8	57.1	57.7	57.9	57.8	58.0	58.6	58.8
Basal	Extra	1	77.2	57.0	56.5	56.9	57.1	57.3	57.6	58.2
Basal	Extra	2	73.5	55.9	56.9	56.6	55.5	55.5	56.2	56.2
Extra	Basal	11	66.4	49.6	50.3	50.2	50.5	51.0	51.5	51.8
Extra	Basal	12	81.0	63.2	64.8	66.6	64.7	65.9	64.5	64.5
Extra	Extra	8	64.6	47.5	47.4	50.4	51.3	51.6	51.8	52.5
Extra	Extra	9	72.9	58.1	58.3	58.5	58.6	58.9	59.0	59.4
		<i>m</i>	72.30	54.48	54.96	55.64	55.52	55.94	56.10	56.45
CALORIC GROUP T										
Basal	Basal	104	68.1	51.6	51.9	52.7	52.8	53.3	53.9	54.6
Basal	Basal	105	68.7	51.8	52.6	53.3	53.4	53.9	54.8	54.8
Basal	Extra	101	64.5	49.7	50.8	50.9	51.0	51.5	51.8	52.2
Basal	Extra	102	67.9	51.9	53.1	54.1	53.4	54.6	55.5	56.2
Extra	Basal	111	63.0	49.1	50.4	51.8	51.8	52.5	53.2	53.8
Extra	Basal	112	61.7	49.0	50.9	51.1	50.7	51.7	51.7	52.1
Extra	Extra	108	66.9	54.1	54.4	54.9	54.9	55.3	55.3	56.1
Extra	Extra	109	79.3	59.5	60.2	60.6	59.4	59.7	60.5	60.6
		<i>m</i>	67.51	52.09	53.04	53.68	53.42	54.06	54.59	55.05
		<i>M</i>	69.39	52.57	53.22	53.49	53.36	53.68	54.01	54.22
		<i>SD</i>	5.85	4.04						

TABLE 450 *continued*

Supplement		Subject	Period					
Protein	Vitamin		R7	R8	R9	R10	R11	R12
CALORIC GROUP Z								
Basal	Basal	122	48.4	48.7	49.0	49.8	50.9	51.6
Basal	Basal	123	51.4	51.7	51.8	52.7	53.4	54.2
Basal	Extra	119	50.0	50.7	50.7	51.6	52.8	53.2
Basal	Extra	120	50.9	51.6	52.2	52.8	53.4	53.7
Extra	Basal	129	53.1	54.3	54.8	55.6	56.4	57.3
Extra	Basal	130	53.0	53.3	54.0	54.7	54.8	55.9
Extra	Extra	126	61.6	62.0	62.4	62.8	63.1	64.4
Extra	Extra	127	50.3	50.6	51.5	52.2	52.4	53.1
		<i>m</i>	52.34	52.86	53.30	54.02	54.65	55.42
CALORIC GROUP L								
Basal	Basal	22	50.8	51.3	51.9	52.7	53.5	54.0
Basal	Basal	23	52.1	53.3	53.9	54.6	55.1	56.2
Basal	Extra	19	52.2	52.7	53.0	54.0	54.9	55.5
Basal	Extra	20	50.9	51.4	52.0	52.9	53.5	54.1
Extra	Basal	29	56.2	57.0	57.8	58.2	58.6	58.8
Extra	Basal	30	55.0	55.7	56.5	56.7	57.2	58.4
Extra	Extra	26	55.9	57.2	57.7	58.6	59.2	60.2
Extra	Extra	27	56.0	56.5	57.1	57.7	57.8	58.6
		<i>m</i>	53.64	54.39	54.99	55.68	56.22	56.98
CALORIC GROUP G								
Basal	Basal	4	51.4	52.4	53.7	55.1	56.3	57.7
Basal	Basal	5	59.3	59.9	60.3	60.8	62.1	62.7
Basal	Extra	1	58.6	59.8	60.3	61.6	62.4	64.0
Basal	Extra	2	56.8	57.6	58.8	60.0	60.9	62.2
Extra	Basal	11	52.9	54.1	55.3	56.4	56.9	57.7
Extra	Basal	12	64.6	64.3	64.3	65.8	65.8	67.4
Extra	Extra	8	53.0	54.3	55.1	56.6	56.8	57.9
Extra	Extra	9	60.2	61.0	61.6	62.6	62.7	64.2
		<i>m</i>	57.10	57.92	58.68	59.86	60.49	61.72
CALORIC GROUP T								
Basal	Basal	104	55.9	56.6	57.4	58.6	59.7	61.1
Basal	Basal	105	56.2	56.8	57.5	58.8	59.9	61.0
Basal	Extra	101	53.3	54.2	54.9	56.2	57.0	58.0
Basal	Extra	102	57.0	58.0	59.0	60.3	60.9	62.2
Extra	Basal	111	54.6	55.7	56.7	58.1	59.1	60.1
Extra	Basal	112	53.0	54.0	55.1	57.0	58.0	59.1
Extra	Extra	108	56.9	57.1	57.7	58.9	59.2	60.2
Extra	Extra	109	61.0	62.0	62.6	64.1	64.4	65.6
		<i>m</i>	55.99	56.80	57.61	59.00	59.78	60.91
		<i>M</i>	54.76	55.49	56.14	57.14	57.75	58.76

TABLE 450 *continued*

Supplement			Period							
Protein	Vitamin	Subject	R13	R14	R15	R16	R17	R18	R19	R20
CALORIC GROUP Z										
Basal	Basal	123	56.8	60.8	63.1	64.5	65.9	67.7	68.0	68.9
Basal	Extra	119	58.3	62.3	65.8	68.1	69.8	70.7	72.0	72.0
Extra	Basal	129	59.4	63.7	66.2	67.4	68.2	68.5	69.4	69.9
Extra	Extra	127	55.8	59.8	62.3	64.4	65.8	66.8	67.0	67.0
CALORIC GROUP L										
Basal	Basal	23	58.9	62.3	64.5	66.9	67.7	68.8	69.3	69.4
Extra	Extra	26	65.9	71.2	73.6	75.0	76.0	76.3	76.2	76.6
CALORIC GROUP G										
Basal	Basal	4	60.2	63.0	64.6	65.7	65.8	66.4	66.4	66.4
Basal	Extra	2	64.8	68.8	71.3	73.3	74.3	75.6	76.0	74.6
CALORIC GROUP T										
Basal	Basal	104	64.0	68.2	69.8	72.6	74.4	75.9	76.0	76.0
Basal	Extra	101	58.4	61.6	64.2	65.7	66.3	66.3	66.6	66.9
Extra	Basal	112	60.8	62.0	63.0	63.5	64.1	64.7	64.5	64.7
Extra	Extra	109	66.9	69.9	72.3	75.4	74.9	76.6	77.2	77.7

TABLE 450 *continued*

Supplement		Subject	Period					
Protein	Vitamin		C12	S24	R12	R20	R33	R58
CALORIC GROUP Z								
Basal	Basal	122	65.4	47.4	51.6		74.0	
Basal	Basal	123	64.7	52.1	54.2	68.9	74.1	
Basal	Extra	119	66.3	49.1	53.2	72.0	70.0	
Basal	Extra	120	70.8	51.6	53.7			
Extra	Basal	129	65.6	52.2	57.3	69.9	73.5	
Extra	Basal	130	66.0	53.6	55.9		76.7	71.4
Extra	Extra	126	83.6	60.6	64.4			
Extra	Extra	127	64.2	49.3	53.1	67.0	70.5	
		<i>m</i>	68.32	51.99	55.42			
CALORIC GROUP L								
Basal	Basal	22	65.1	49.4	54.0		73.7	66.4
Basal	Basal	23	69.2	51.4	56.2	69.4	68.5	72.0
Basal	Extra	19	70.6	50.4	55.5		84.7	
Basal	Extra	20	64.7	48.0	54.1			
Extra	Basal	29	71.1	53.5	58.8		84.6	
Extra	Basal	30	68.1	52.4	58.4		75.9	
Extra	Extra	26	71.3	53.1	60.2	76.6	80.5	
Extra	Extra	27	75.2	55.7	58.6		75.8	
		<i>m</i>	69.41	51.74	56.98			
CALORIC GROUP G								
Basal	Basal	4	62.0	47.4	57.7	66.4	65.7	
Basal	Basal	5	80.8	57.1	62.7		79.6	
Basal	Extra	1	77.2	57.0	64.0		84.6	
Basal	Extra	2	73.5	55.9	62.2	74.6	78.6	71.3
Extra	Basal	11	66.4	49.6	57.7		62.2	
Extra	Basal	12	81.0	63.2	67.4			
Extra	Extra	8	64.6	47.5	57.9			
Extra	Extra	9	72.9	58.1	64.2		93.0	
		<i>m</i>	72.30	54.48	61.72			
CALORIC GROUP T								
Basal	Basal	104	68.1	51.6	61.1	76.0		
Basal	Basal	105	68.7	51.8	61.0			
Basal	Extra	101	64.5	49.7	58.0	66.9		
Basal	Extra	102	67.9	51.9	62.2			
Extra	Basal	111	63.0	49.1	60.1			
Extra	Basal	112	61.7	49.0	59.1	64.7	67.4	66.0
Extra	Extra	108	66.9	54.1	60.2			
Extra	Extra	109	79.3	59.5	65.6	77.7	78.0	80.2
		<i>m</i>	67.51	52.09	60.91			
		<i>M</i>	69.39	52.57	58.76			
		<i>SD</i>	5.85	4.04				

TABLE 451

BODY DIMENSIONS DURING THE CONTROL PERIOD, in cm.: *standing height* (column 1), *sitting height* (column 2), *bi-cristal diameter* (column 3), *bi-trochanteric diameter* (column 4), *bi-acromial diameter* (column 5), *chest, anteroposterior diameter* (column 6), *chest, transverse diameter* (column 7), *chest, circumference* (column 8).

Supplement		Subject	Period							
Protein	Vitamin		1	2	3	4	5	6	7	8
CALORIC GROUP Z										
Basal	Basal	122	167.5	90.3	24.1	29.6	36.8	18.0	25.4	90.0
Basal	Basal	123	181.0	96.2	27.6	31.3	37.6	16.9	25.7	87.1
Basal	Extra	119	176.9	89.6	27.4	32.0	39.8	17.5	26.9	87.9
Basal	Extra	120	172.3	91.5	27.3	30.8	38.1	20.4	25.6	90.0
Extra	Basal	129	187.5	94.6	30.6	33.1	37.5	16.9	26.6	82.0
Extra	Basal	130	189.2	93.8	27.2	28.4	36.6	19.9	26.5	87.8
Extra	Extra	126	185.3	97.9	30.2	35.4	42.4	18.6	29.8	92.6
Extra	Extra	127	180.1	91.6	25.8	30.0	35.7	19.6	25.4	87.8
		<i>m</i>	179.98	93.19	27.52	31.32	38.06	18.48	26.49	88.15
CALORIC GROUP L										
Basal	Basal	22	177.5	94.4	27.3	32.3	40.8	20.0	27.0	89.0
Basal	Basal	23	177.7	91.0	28.6	30.4	38.8	17.0	25.4	88.1
Basal	Extra	19	170.4	88.8	27.5	33.0	37.6	17.7	27.2	91.2
Basal	Extra	20	175.8	94.6	26.9	30.6	38.7	19.4	26.9	87.4
Extra	Basal	29	178.6	94.0	28.8	34.1	38.8	19.0	28.7	93.4
Extra	Basal	30	180.8	91.5	29.0	34.8	38.4	18.7	26.8	91.7
Extra	Extra	26	180.5	97.7	28.0	33.4	39.3	16.4	28.3	89.1
Extra	Extra	27	177.8	93.6	27.1	32.8	36.6	20.4	28.6	93.5
		<i>m</i>	177.39	93.20	27.90	32.68	38.62	18.58	27.36	90.42
CALORIC GROUP G										
Basal	Basal	4	174.3	89.0	25.9	28.9	38.0	15.8	26.1	83.6
Basal	Basal	5	178.4	91.4	30.1	31.6	38.8	19.0	28.4	98.7
Basal	Extra	1	186.0	97.6	28.5	32.8	37.0	17.5	28.8	89.0
Basal	Extra	2	177.9	90.9	27.9	32.6	36.0	19.8	28.4	92.2
Extra	Basal	11	178.0	94.0	28.0	31.2	37.1	18.8	28.1	91.1
Extra	Basal	12	191.9	98.2	30.1	35.1	41.0	18.1	27.7	91.3
Extra	Extra	8	168.0	89.4	26.4	30.5	39.1	17.8	27.5	90.7
Extra	Extra	9	188.3	97.1	30.9	31.9	40.4	19.2	25.8	88.2
		<i>m</i>	180.35	93.45	28.48	31.82	38.42	18.25	27.60	90.60
CALORIC GROUP T										
Basal	Basal	104	176.2	90.1	28.7	31.4	40.2	19.6	27.1	89.6
Basal	Basal	105	175.7	90.5	28.4	30.5	38.3	18.9	24.6	85.7
Basal	Extra	101	177.6	94.0	27.5	31.5	39.5	19.2	25.4	86.5
Basal	Extra	102	178.7	93.4	27.7	32.5	37.5	19.3	26.6	84.6
Extra	Basal	111	178.4	94.0	28.6	31.5	35.8	19.6	25.6	87.3
Extra	Basal	112	176.0	89.5	25.0	29.1	35.4	17.7	25.5	85.6
Extra	Extra	108	170.5	90.3	26.8	31.6	36.6	19.2	27.1	90.9
Extra	Extra	109	187.1	98.3	31.1	33.6	39.8	19.0	27.0	93.3
		<i>m</i>	177.52	92.15	27.98	31.46	37.89	19.06	26.11	87.94
		<i>M</i>	178.81	93.09	27.97	31.82	38.25	18.59	26.89	89.28
		<i>SD</i>	5.77	3.00	1.36	1.75	1.70	1.02	1.28	3.33

TABLE 452
BI-HUMERAL DIAMETER, in cm.

Supplement		Subject	Period					R20
Protein	Vitamin		C	S12	S24	dS24	△R12	
CALORIC GROUP Z								
Basal	Basal	122	43.5	40.0	38.2	-5.3	1.9	44.8
Basal	Basal	123	43.2	40.0	39.0	-4.2	1.0	
Basal	Extra	119	44.1	41.2	39.7	-4.4	1.3	
Basal	Extra	120	43.2	40.1	39.3	-3.9	0.5	42.0
Extra	Basal	129	42.2	40.0	39.6	-2.6	0.1	
Extra	Basal	130	41.4	38.7	38.4	-3.0	0.2	
Extra	Extra	126	47.8	44.4	43.3	-4.5	0.2	41.6
Extra	Extra	127	41.7	37.5	37.0	-4.7	0.6	
		<i>m</i>	43.39	40.24	39.31	-4.08	0.72	
CALORIC GROUP L								
Basal	Basal	22	44.7	41.6	40.9	-3.8	0.9	43.4
Basal	Basal	23	43.2	40.0	39.4	-3.8	1.0	
Basal	Extra	19	42.7	40.6	39.5	-3.2	0.7	
Basal	Extra	20	43.4	41.2	39.4	-4.0	1.6	46.2
Extra	Basal	29	44.0	40.6	40.2	-3.8	1.3	
Extra	Basal	30	44.0	40.9	40.6	-3.4	1.1	
Extra	Extra	26	45.8	42.8	41.0	-4.8	1.8	46.2
Extra	Extra	27	43.6	39.4	39.2	-4.4	0.2	
		<i>m</i>	43.92	40.89	40.02	-3.90	1.08	
CALORIC GROUP C								
Basal	Basal	4	43.4	38.6	38.2	-5.2	3.1	44.2
Basal	Basal	5	45.6	42.6	41.5	-4.1	1.3	42.9
Basal	Extra	1	43.7	41.6	40.2	-3.5	1.2	
Basal	Extra	2	43.7	39.5	38.9	-4.8	1.7	
Extra	Basal	11	43.1	39.6	39.4	-3.7	1.4	42.9
Extra	Basal	12	46.1	42.4	41.5	-4.6	1.7	
Extra	Extra	8	43.2	39.5	39.4	-3.8	1.5	
Extra	Extra	9	43.7	40.0	40.6	-3.1	0.8	42.9
		<i>m</i>	44.06	40.48	39.96	-4.10	1.59	
CALORIC GROUP T								
Basal	Basal	104	44.8	41.7	41.0	-3.8	1.6	46.3
Basal	Basal	105	43.6	39.3	38.5	-5.1	2.7	42.8
Basal	Extra	101	43.0	41.5	40.5	-2.5	0.5	
Basal	Extra	102	42.7	39.0	37.8	-4.9	3.6	
Extra	Basal	111	41.6	39.5	38.1	-3.5	1.8	42.7
Extra	Basal	112	41.9	38.7	37.8	-4.1	3.6	
Extra	Extra	108	42.1	39.1	37.8	-4.3	2.5	
Extra	Extra	109	44.7	41.3	40.4	-4.3	2.9	44.8
		<i>m</i>	43.05	40.01	38.99	-4.06	2.40	44.8
		<i>M</i>	43.60	40.40	39.57	-4.03	1.45	
		<i>SD</i>	1.39	1.46	1.36			

TABLE 453
CIRCUMFERENCE OF THE UPPER ARM, in cm.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	△R12	R20
CALORIC GROUP Z								
Basal	Basal	122	30.2	24.6	21.8	-8.4	2.0	28.6
Basal	Basal	123	28.6	24.0	21.5	-7.1	1.3	
Basal	Extra	119	26.5	21.7	20.2	-6.3	0.9	26.8
Basal	Extra	120	30.8	25.4	22.8	-8.0	0.9	24.6
Extra	Basal	129	24.6	20.7	19.5	-5.1	1.1	
Extra	Basal	130	26.0	22.4	20.7	-5.3	0.9	28.4
Extra	Extra	126	30.5	25.1	22.4	-8.1	0.5	
Extra	Extra	127	28.8	24.0	22.2	-6.6	1.4	
		<i>m</i>	28.25	23.49	21.39	-6.86	1.12	
CALORIC GROUP L								
Basal	Basal	22	29.3	24.2	22.0	-7.3	2.1	29.4
Basal	Basal	23	28.7	23.4	21.9	-6.8	1.4	
Basal	Extra	19	32.0	25.2	22.0	-10.0	2.5	29.2
Basal	Extra	20	28.2	23.5	20.8	-7.4	2.7	
Extra	Basal	29	28.3	22.3	21.2	-7.1	2.0	29.2
Extra	Basal	30	28.4	23.5	21.5	-6.9	2.1	
Extra	Extra	26	28.6	23.8	21.6	-7.0	2.3	
Extra	Extra	27	30.2	24.6	22.6	-7.6	0.4	
		<i>m</i>	29.21	23.81	21.70	-7.51	1.94	
CALORIC GROUP G								
Basal	Basal	4	26.7	22.4	21.0	-5.7	3.4	27.6
Basal	Basal	5	32.4	26.2	24.0	-8.4	2.2	25.7
Basal	Extra	1	30.8	24.9	22.4	-8.4	2.4	
Basal	Extra	2	27.4	21.9	20.9	-6.5	0.9	
Extra	Basal	11	27.0	22.5	20.9	-6.1	2.2	27.7
Extra	Basal	12	29.8	23.7	21.1	-8.7	2.2	
Extra	Extra	8	27.5	21.5	20.2	-7.3	3.8	27.2
Extra	Extra	9	28.3	22.7	21.7	-6.6	2.9	
		<i>m</i>	28.74	23.22	21.53	-7.21	2.50	
CALORIC GROUP T								
Basal	Basal	104	26.9	22.5	21.1	-5.8	2.5	29.2
Basal	Basal	105	29.0	23.9	21.8	-7.2	3.1	28.4
Basal	Extra	101	27.8	23.4	20.4	-7.4	4.0	
Basal	Extra	102	28.0	23.2	22.2	-5.8	2.6	27.7
Extra	Basal	111	25.9	21.4	20.0	-5.9	2.8	
Extra	Basal	112	26.6	21.6	21.5	-5.1	3.2	27.2
Extra	Extra	108	28.8	23.7	22.3	-6.5	2.9	
Extra	Extra	109	29.1	23.8	22.3	-6.8	1.8	
		<i>m</i>	27.76	22.94	21.45	-6.31	2.86	
		<i>M</i>	28.49	23.37	21.52	-6.97	2.11	
		<i>SD</i>	1.78	1.32	0.94	1.12	0.94	

TABLE 454
CIRCUMFERENCE OF THE THIGH, in cm.

Supplement		Subject	Period					R20
Protein	Vitamin		C	S12	S24	dS24	△R12	
CALORIC GROUP Z								
Basal	Basal	122	49.4	41.8	38.4	-11.0	2.1	47.4
Basal	Basal	123	45.7	40.6	38.8	-6.9	0.4	
Basal	Extra	119	45.0	39.1	36.0	-9.0	2.4	
Basal	Extra	120	49.5	42.3	39.5	-10.0	1.3	44.8
Extra	Basal	129	43.5	38.1	36.5	-7.0	2.3	
Extra	Basal	130	45.4	41.0	37.6	-7.8	2.2	
Extra	Extra	126	53.0	45.0	40.1	-12.9	1.8	43.8
Extra	Extra	127	44.0	36.7	34.6	-9.4	2.4	
		<i>m</i>	46.94	40.58	37.69	-9.25	1.86	
CALORIC GROUP L								
Basal	Basal	22	44.3	37.3	35.3	-9.0	3.5	48.6
Basal	Basal	23	49.5	42.0	39.5	-10.0	3.5	
Basal	Extra	19	52.6	42.6	39.4	-13.2	2.9	
Basal	Extra	20	44.7	38.2	34.4	-10.3	5.5	50.4
Extra	Basal	29	47.8	39.6	39.5	-8.3	1.4	
Extra	Basal	30	44.2	38.1	36.8	-7.4	1.3	
Extra	Extra	26	49.7	41.6	39.2	-10.5	4.2	50.4
Extra	Extra	27	51.6	43.8	40.4	-11.2	3.4	
		<i>m</i>	48.05	40.40	38.06	-9.99	3.21	
CALORIC GROUP G								
Basal	Basal	4	44.4	37.7	36.1	-8.3	5.2	47.0
Basal	Basal	5	51.8	44.5	40.8	-11.0	3.2	
Basal	Extra	1	50.0	43.6	39.8	-10.2	3.3	
Basal	Extra	2	47.5	41.3	40.0	-7.5	1.9	47.9
Extra	Basal	11	44.1	39.7	37.2	-6.9	3.2	
Extra	Basal	12	47.7	41.2	38.0	-9.7	3.9	
Extra	Extra	8	46.8	37.1	35.8	-11.0	6.5	47.9
Extra	Extra	9	47.1	38.7	38.2	-8.9	2.5	
		<i>m</i>	47.43	40.48	38.24	-9.19	3.71	
CALORIC GROUP T								
Basal	Basal	104	46.2	39.1	37.2	-9.0	4.4	48.2
Basal	Basal	105	46.1	39.2	37.9	-8.2	3.9	
Basal	Extra	101	43.4	39.5	35.6	-7.8	4.7	
Basal	Extra	102	48.6	41.2	38.3	-10.3	5.9	44.0
Extra	Basal	111	44.4	37.6	35.7	-8.7	5.7	
Extra	Basal	112	43.4	38.7	37.3	-6.1	5.4	
Extra	Extra	108	47.4	40.1	39.4	-8.0	3.4	44.8
Extra	Extra	109	48.7	42.7	41.5	-7.2	2.1	
		<i>m</i>	46.02	39.76	37.86	-8.16	4.44	
		<i>M</i>	47.11	40.30	37.96	-9.15	3.31	48.3
		<i>SD</i>	2.85	2.26	1.90	1.74	1.53	

TABLE 455
CIRCUMFERENCE OF THE CALF, in cm.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	△R12	R20
CALORIC GROUP Z								
Basal	Basal	122	38.4	33.8	32.3	-6.1	0.7	38.2
Basal	Basal	123	37.5	35.1	34.0	-3.5	0.5	
Basal	Extra	119	36.6	33.4	31.8	-4.8	1.2	37.4
Basal	Extra	120	38.3	35.1	33.0	-5.3	0.6	38.6
Extra	Basal	129	38.1	34.9	34.3	-3.8	1.4	
Extra	Basal	130	36.6	33.4	31.7	-4.9	1.1	37.6
Extra	Extra	126	41.4	38.0	35.0	-6.4	1.2	
Extra	Extra	127	37.0	33.6	32.2	-4.8	1.5	
		<i>m</i>	37.99	34.66	33.04	-4.95	1.02	
CALORIC GROUP L								
Basal	Basal	22	36.7	33.1	31.4	-5.3	1.3	37.1
Basal	Basal	23	38.0	34.1	33.4	-4.6	0.8	
Basal	Extra	19	39.0	35.2	33.4	-5.6	1.2	40.0
Basal	Extra	20	34.1	31.0	28.7	-5.4	2.5	
Extra	Basal	29	37.7	33.0	32.4	-5.3	0.8	40.0
Extra	Basal	30	35.7	33.2	31.8	-3.9	1.4	
Extra	Extra	26	38.6	34.8	33.4	-5.2	1.9	40.0
Extra	Extra	27	40.1	36.4	35.0	-5.1	0.6	
		<i>m</i>	37.49	33.85	32.44	-5.05	1.31	
CALORIC GROUP G								
Basal	Basal	4	35.8	32.4	31.5	-4.3	2.1	36.7
Basal	Basal	5	39.6	36.2	34.4	-5.2	0.4	39.7
Basal	Extra	1	38.4	34.6	32.8	-5.6	1.6	
Basal	Extra	2	39.7	36.2	35.6	-4.1	0.8	39.7
Extra	Basal	11	33.8	31.1	29.8	-4.0	1.6	
Extra	Basal	12	38.7	37.0	35.4	-3.3	0.1	35.0
Extra	Extra	8	37.3	32.5	31.8	-5.5	3.4	
Extra	Extra	9	39.8	36.2	35.4	-4.4	1.1	37.8
		<i>m</i>	37.89	34.52	33.34	-4.55	1.39	
CALORIC GROUP T								
Basal	Basal	104	38.1	33.9	32.3	-5.8	2.0	39.0
Basal	Basal	105	38.4	35.2	34.3	-4.1	1.1	36.6
Basal	Extra	101	36.3	33.3	32.0	-4.3	1.8	
Basal	Extra	102	38.6	35.2	32.8	-5.8	3.2	35.0
Extra	Basal	111	38.0	34.4	33.8	-4.2	3.1	
Extra	Basal	112	34.6	31.8	31.2	-3.4	2.2	37.8
Extra	Extra	108	37.6	34.2	34.3	-3.3	1.4	
Extra	Extra	109	38.5	36.4	35.1	-3.4	0.9	
		<i>m</i>	37.51	34.30	33.22	-4.29	1.96	
		<i>M</i>	37.72	34.33	33.01	-4.71	1.42	
		<i>SD</i>	1.71	1.68	1.66	0.78	0.81	

TABLE 456
ABDOMINAL CIRCUMFERENCE, in cm.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	ΔR12	R20
CALORIC GROUP Z								
Basal	Basal	122	73.4	66.8	65.1	-8.3	5.3	82.3
Basal	Basal	123	75.4	70.5	70.8	-4.6	2.7	
Basal	Extra	119	77.6	70.2	69.4	-8.2	4.2	85.2
Basal	Extra	120	80.9	68.7	67.4	-13.5	2.8	84.1
Extra	Basal	129	74.6	69.4	71.1	-3.5	5.0	
Extra	Basal	130	74.9	70.7	71.6	-3.3	-0.4	80.1
Extra	Extra	126	84.0	75.9	70.7	-13.3	4.7	
Extra	Extra	127	72.4	66.9	66.5	-5.9	2.7	
		<i>m</i>	76.65	69.88	69.07	-7.58	3.38	
CALORIC GROUP L								
Basal	Basal	22	76.0	70.3	70.2	-5.8	2.3	81.5
Basal	Basal	23	75.8	70.0	70.6	-5.2	3.0	
Basal	Extra	19	80.2	74.8	73.8	-6.4	2.2	82.8
Basal	Extra	20	75.3	68.7	68.3	-7.0	3.7	
Extra	Basal	29	78.9	72.3	73.2	-5.7	1.4	82.8
Extra	Basal	30	76.3	70.6	70.4	-5.9	4.9	
Extra	Extra	26	75.6	67.8	65.3	-10.3	7.0	
Extra	Extra	27	78.6	69.0	66.4	-12.2	5.0	
		<i>m</i>	77.09	70.43	69.78	-7.31	3.69	
CALORIC GROUP G								
Basal	Basal	4	73.7	67.2	67.1	-6.6	9.3	82.2
Basal	Basal	5	91.8	78.8	77.1	-14.7	3.1	88.2
Basal	Extra	1	78.6	75.8	73.1	-5.5	4.7	
Basal	Extra	2	82.7	72.0	71.6	-11.1	6.0	
Extra	Basal	11	83.9	72.4	70.8	-13.1	6.3	78.6
Extra	Basal	12	80.2	75.8	76.0	-4.2	2.4	
Extra	Extra	8	78.2	70.6	70.0	-8.2	7.0	88.5
Extra	Extra	9	78.2	76.9	77.7	- .5	3.2	
		<i>m</i>	80.91	73.69	72.92	-7.99	5.25	
CALORIC GROUP T								
Basal	Basal	104	77.7	70.0	71.7	-6.0	6.6	87.9
Basal	Basal	105	77.1	70.0	72.4	-4.7	7.3	78.0
Basal	Extra	101	74.6	65.0	66.6	-8.0	5.9	
Basal	Extra	102	75.2	70.6	67.6	-7.6	9.3	78.6
Extra	Basal	111	76.0	72.6	71.7	-4.3	7.7	
Extra	Basal	112	71.9	67.6	65.7	-6.2	9.1	
Extra	Extra	108	79.9	74.3	75.6	-4.3	4.2	88.5
Extra	Extra	109	88.8	77.3	76.8	-12.0	3.3	
		<i>m</i>	77.65	70.92	71.01	-6.64	6.68	
		<i>M</i>	78.08	71.23	70.70	-7.38	4.75	
		<i>SD</i>	4.43	3.43	3.54	3.48	2.38	

TABLE 457

CALCULATED BODY FAT, in kg., not corrected for the changes in hydration
and the relative mass of the bones.

Supplement		Subject	Period					R20
Protein	Vitamin		C	S12	S24	dS24	△R12	
CALORIC GROUP Z								
Basal	Basal	122	4.2	0.0	0.0	-4.2	0.0	12.1
Basal	Basal	123	6.7	2.3	2.0	-4.7	0.8	
Basal	Extra	119	11.2	6.2	1.9	-9.3	3.6	14.7
Basal	Extra	120	14.1	8.8	3.4	-10.7	1.7	12.0
Extra	Basal	129	6.1	1.3	2.0	-4.1	2.9	
Extra	Basal	130	13.8	8.8	9.4	-4.4	-1.9	12.8
Extra	Extra	126	17.5	8.4	5.6	-11.9	0.5	
Extra	Extra	127	5.6	2.9	2.3	-3.3	1.4	
		<i>m</i>	9.90	4.84	3.32	-6.58	1.12	
CALORIC GROUP L								
Basal	Basal	22	9.5	3.5	3.0	-6.5	3.1	12.5
Basal	Basal	23	12.1	5.2	3.4	-8.7	3.0	
Basal	Extra	19	9.0	2.7	2.0	-7.0	2.2	13.0
Basal	Extra	20	6.7	3.6	0.6	-6.1	3.5	
Extra	Basal	29	8.6	5.1	6.2	-2.4	0.5	13.0
Extra	Basal	30	8.3	6.5	3.5	-4.8	4.1	
Extra	Extra	26	6.6	0.5	1.5	-5.1	2.5	13.0
Extra	Extra	27	11.4	2.5	1.0	-10.4	2.8	
		<i>m</i>	9.02	3.70	2.65	-6.37	2.71	
CALORIC GROUP G								
Basal	Basal	4	4.5	1.4	0.0	-4.5	5.7	10.7
Basal	Basal	5	19.4	9.5	5.1	-14.3	3.0	15.8
Basal	Extra	1	10.8	3.6	2.7	-8.1	2.8	
Basal	Extra	2	12.5	7.7	2.7	-9.8	4.4	15.8
Extra	Basal	11	11.3	6.1	2.3	-9.0	4.3	
Extra	Basal	12	11.4	4.8	4.0	-7.4	1.8	15.8
Extra	Extra	8	5.4	0.0	0.0	-5.4	4.4	
Extra	Extra	9	8.2	0.6	2.4	-5.8	3.1	15.8
		<i>m</i>	10.44	4.21	2.40	-8.04	3.69	
CALORIC GROUP T								
Basal	Basal	104	5.0	4.2	2.4	-2.6	4.0	14.0
Basal	Basal	105	7.3	3.9	0.7	-6.6	7.2	15.1
Basal	Extra	101	11.6	4.0	3.7	-7.9	7.8	
Basal	Extra	102	13.6	7.4	3.6	-10.0	7.6	10.4
Extra	Basal	111	9.0	2.9	3.0	-6.0	6.9	
Extra	Basal	112	6.1	2.8	3.2	-2.9	5.8	21.7
Extra	Extra	108	6.9	3.8	2.0	-4.9	4.3	
Extra	Extra	109	20.6	15.2	11.9	-8.7	3.4	21.7
		<i>m</i>	10.01	5.52	3.81	-6.20	5.88	
		<i>M</i>	9.84	4.57	3.04	-6.80	3.35	
		<i>SD</i>	4.18	3.29	2.51	2.90	2.24	

TABLE 458

POSITION OF THE MAJOR ANATOMICAL AXIS OF THE HEART, measured in degrees of angle from the horizontal, as estimated from roentgenkymograms.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	R12	R20
CALORIC GROUP Z								
Basal	Basal	122	55.6	60.8	53.0	—2.6		
Basal	Basal	123	47.0	56.2	50.5	3.5	50.4	38.4
Basal	Extra	119	42.8	52.8	49.8	7.0	50.0	37.0
Basal	Extra	120	51.5	60.7	59.6	8.1		
Extra	Basal	129	54.3	61.6	58.0	3.7	56.0	52.2
Extra	Basal	130	45.7	57.0	53.8	8.1		
Extra	Extra	126	56.2	55.6	59.5	3.3		
Extra	Extra	127	51.3	64.8	58.6	7.3	52.0	50.4
		<i>m</i>	50.55	58.69	55.35	4.80		
CALORIC GROUP L								
Basal	Basal	22	51.8	62.5	57.5	5.7		
Basal	Basal	23	45.6	61.5	55.8	10.2	51.6	51.2
Basal	Extra	19	43.2	48.8	49.6	6.4		
Basal	Extra	20	51.0	55.5	58.0	7.0		
Extra	Basal	29	41.7	49.5	48.8	7.1		
Extra	Basal	30	50.0	57.0	52.0	2.0		
Extra	Extra	26	49.2	56.0	50.8	1.6	45.0	39.6
Extra	Extra	27	50.0	58.8	60.8	10.8		
		<i>m</i>	47.81	56.20	54.16	6.35		
CALORIC GROUP G								
Basal	Basal	4	37.1	53.6	38.7	1.6	35.8	37.0
Basal	Basal	5	48.5	58.3	58.8	10.3		
Basal	Extra	1	51.2	54.0	51.2	0.0		
Basal	Extra	2	43.5	55.8	48.6	5.1	47.5	44.6
Extra	Basal	11	50.6	58.3	60.0	9.4		
Extra	Basal	12	55.3	58.6	60.4	5.1		
Extra	Extra	8	47.7	58.5	59.0	11.3		
Extra	Extra	9	49.0	60.0	61.5	12.5		
		<i>m</i>	47.86	57.14	54.77	6.91		
CALORIC GROUP T								
Basal	Basal	104	46.8	54.2	48.0	1.2	43.6	39.6
Basal	Basal	105	55.0	62.7	60.0	5.0		
Basal	Extra	101	52.5	62.0	52.8	0.3	56.2	54.7
Basal	Extra	102	52.0	58.0	57.0	5.0		
Extra	Basal	111	63.2	69.8	70.7	7.5		
Extra	Basal	112	47.4	50.8	52.5	5.1	48.4	47.5
Extra	Extra	108	48.8	60.8	56.3	7.5		
Extra	Extra	109	44.8	59.3	50.4	5.6	51.7	46.6
		<i>m</i>	51.31	59.70	55.96	4.65		
		<i>M</i>	49.38	57.93	55.06	5.68		
		<i>SD</i>	5.08		5.86			

TABLE 459

TRANSVERSE DIAMETER OF THE HEART, in cm., in systole. Measurements made on roentgenkymograms and corrected for triangular distortion.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	11.04	10.41	—0.63		
Basal	Basal	123	11.28	10.20	—1.08	11.27	12.77
Basal	Extra	119	11.66	9.75	—1.91	10.34	12.32
Basal	Extra	120	11.48	9.58	—1.90		
Extra	Basal	129	10.51	8.96	—1.55	10.24	10.21
Extra	Basal	130	12.32	10.63	—1.69		
Extra	Extra	126	12.07	11.33	—0.74		
Extra	Extra	127	10.69	9.19	—1.50	10.25	10.63
		<i>m</i>	11.381	10.006	—1.375		
CALORIC GROUP L							
Basal	Basal	22	11.73	10.10	—1.63		
Basal	Basal	23	10.76	8.84	—1.92	9.81	10.50
Basal	Extra	19	12.18	11.68	—0.50		
Basal	Extra	20	11.94	9.98	—1.96		
Extra	Basal	29	13.28	11.87	—1.41		
Extra	Basal	30	11.04	10.29	—0.75		
Extra	Extra	26	12.00	11.05	—0.95	12.15	12.93
Extra	Extra	27	13.03	10.15	—2.88		
		<i>m</i>	11.995	10.495	—1.500		
CALORIC GROUP G							
Basal	Basal	4	13.30	10.68	—2.62	12.62	13.96
Basal	Basal	5	12.51	9.79	—2.72		
Basal	Extra	1	12.02	11.26	—0.76		
Basal	Extra	2	12.42	12.02	—0.40	12.34	12.57
Extra	Basal	11	12.27	9.52	—2.75		
Extra	Basal	12	11.73	10.86	—0.87		
Extra	Extra	8	13.07	10.53	—2.54		
Extra	Extra	9	11.53	9.10	—2.43		
		<i>m</i>	12.356	10.470	—1.886		
CALORIC GROUP T							
Basal	Basal	104	10.91	10.67	—0.24	11.90	12.26
Basal	Basal	105	10.42	9.44	—0.98		
Basal	Extra	101	10.22	9.08	—1.14	10.08	10.35
Basal	Extra	102	11.65	9.78	—1.87		
Extra	Basal	111	10.10	8.74	—1.36		
Extra	Basal	112	11.11	9.17	—1.94	10.63	11.02
Extra	Extra	108	12.06	11.07	—0.99		
Extra	Extra	109	12.41	10.74	—1.67	11.39	11.96
		<i>m</i>	11.110	9.836	—1.274		
		<i>M</i>	11.711	10.202	—1.509		
		<i>SD</i>	0.873	0.912			

TABLE 460

HEART SIZE, in ventricular systole. Measured as the area, in sq. cm., of the projected kymographic silhouette, corrected for triangular distortion.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	106.7	100.7	—6.0		
Basal	Basal	123	110.4	102.7	—7.7	112.1	116.4
Basal	Extra	119	105.1	87.5	—17.6	98.4	105.5
Basal	Extra	120	113.5	97.7	—15.8		
Extra	Basal	129	106.0	83.2	—22.8	97.8	98.0
Extra	Basal	130	113.6	99.1	—14.5		
Extra	Extra	126	133.5	126.3	—7.2		
Extra	Extra	127	96.8	84.1	—12.7	94.4	99.9
		<i>m</i>	110.70	97.66	—13.04		
CALORIC GROUP L							
Basal	Basal	22	113.3	94.8	—18.5		
Basal	Basal	23	101.3	86.1	—15.2	91.4	98.8
Basal	Extra	19	111.6	109.2	—2.4		
Basal	Extra	20	113.5	103.2	—10.3		
Extra	Basal	29	140.6	122.3	—18.3		
Extra	Basal	30	102.7	94.5	—8.2		
Extra	Extra	26	118.7	112.6	—6.1	118.7	131.3
Extra	Extra	27	127.5	107.0	—20.5		
		<i>m</i>	116.15	103.71	—12.44		
CALORIC GROUP G							
Basal	Basal	4	121.4	97.9	—23.5	116.4	126.7
Basal	Basal	5	123.7	109.0	—14.7		
Basal	Extra	1	119.1	108.2	—10.9		
Basal	Extra	2	120.4	111.5	—8.9	121.3	118.4
Extra	Basal	11	114.4	95.3	—19.1		
Extra	Basal	12	121.1	115.4	—5.7		
Extra	Extra	8	134.5	107.0	—27.5		
Extra	Extra	9	115.4	97.1	—18.3		
		<i>m</i>	121.25	105.18	—16.07		
CALORIC GROUP T							
Basal	Basal	104	98.7	87.1	—11.6	101.0	107.4
Basal	Basal	105	110.4	96.7	—13.7		
Basal	Extra	101	102.4	86.2	—16.2	102.8	107.2
Basal	Extra	102	126.3	99.9	—26.4		
Extra	Basal	111	102.0	87.2	—14.8		
Extra	Basal	112	100.8	86.3	—14.5	97.3	103.7
Extra	Extra	108	115.8	111.7	—4.1		
Extra	Extra	109	122.8	108.4	—14.4	114.3	121.3
		<i>m</i>	109.90	95.44	—14.46		
		<i>M</i>	114.50	100.50	—14.00		
		<i>SD</i>	10.93	11.31			

TABLE 461

HEART SIZE, in ventricular systole. Measured as the total volume, in cc., calculated from the projected kymographic silhouette, corrected for triangular distortion.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	R12	R20
CALORIC GROUP Z								
Basal	Basal	122	558.3	539.6	513.4	—44.9		
Basal	Basal	123	586.8	530.2	528.7	—58.1	599.9	633.7
Basal	Extra	119	546.2	470.6	419.0	—127.2	496.6	549.2
Basal	Extra	120	610.8	444.8	491.5	—119.3		
Extra	Basal	129	553.0	417.0	389.4	—163.6	492.3	493.7
Extra	Basal	130	611.5	579.7	501.8	—109.7		
Extra	Extra	126	773.2	603.7	713.1	—60.1		
Extra	Extra	127	485.0	387.9	395.5	—89.5	467.8	507.6
		<i>m</i>	590.60	496.69	494.05	—96.55		
CALORIC GROUP L								
Basal	Basal	22	609.2	476.9	470.6	—138.6		
Basal	Basal	23	518.0	409.4	409.4	—108.6	446.2	499.5
Basal	Extra	19	596.0	673.5	577.5	—18.5		
Basal	Extra	20	610.8	551.4	532.3	—78.5		
Extra	Basal	29	833.3	644.8	680.9	—152.4		
Extra	Basal	30	528.7	431.1	468.5	—60.2		
Extra	Extra	26	652.0	615.5	603.7	—48.3	652.0	754.6
Extra	Extra	27	723.0	564.0	560.6	—162.4		
		<i>m</i>	633.88	545.82	537.94	—95.94		
CALORIC GROUP G								
Basal	Basal	4	673.5	526.4	493.0	—180.5	633.7	716.4
Basal	Basal	5	692.0	690.5	576.0	—116.0		
Basal	Extra	1	655.0	688.9	569.8	—85.2		
Basal	Extra	2	665.4	648.8	595.3	—70.1	672.7	649.5
Extra	Basal	11	617.9	526.4	473.9	—144.0		
Extra	Basal	12	671.0	671.9	625.8	—45.2		
Extra	Extra	8	781.6	591.4	560.6	—221.0		
Extra	Extra	9	625.8	513.4	487.2	—138.6		
		<i>m</i>	672.78	607.21	547.70	—125.08		
CALORIC GROUP T								
Basal	Basal	104	498.8	439.8	416.2	—82.6	515.8	563.7
Basal	Basal	105	586.8	527.1	484.3	—102.5		
Basal	Extra	101	526.4	375.6	410.0	—116.4	529.4	562.1
Basal	Extra	102	713.1	526.4	507.6	—205.5		
Extra	Basal	111	523.4	439.1	417.0	—106.4		
Extra	Basal	112	514.1	439.1	410.8	—103.3	488.7	536.0
Extra	Extra	108	628.9	602.2	596.8	—32.1		
Extra	Extra	109	684.8	611.5	571.4	—113.4	617.1	672.7
		<i>m</i>	584.54	495.10	476.76	—107.78		
		<i>M</i>	620.45	536.21	514.11	—106.34		
		<i>SD</i>	86.41		84.05			

TABLE 462
HEMATOCRIT, as cell volume per 100 cc. of whole venous blood.

Supplement			Period				
Protein	Vitamin	Subject	C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	46.1	39.0	—7.1	39.2	
Basal	Basal	123	43.1	29.7	—13.4		
Basal	Extra	119	46.0	40.8	—5.2	38.6	
Basal	Extra	120	48.9	43.7	—5.2	43.0	
Extra	Basal	129	47.3	38.9	—8.4		
Extra	Basal	130	46.0	33.1	—12.9	37.3	47.3
Extra	Extra	126	44.8	37.5	—7.3		
Extra	Extra	127	49.7	38.3	—11.4		
		<i>m</i>	46.49	37.62	—8.87		
CALORIC GROUP L							
Basal	Basal	22	46.1	35.7	—10.4	39.8	
Basal	Basal	23	46.5	38.2	—8.3		
Basal	Extra	19	47.6	32.0	—15.6		
Basal	Extra	20	47.9	34.2	—13.7	36.4	
Extra	Basal	29	46.4	32.6	—13.8	41.8	
Extra	Basal	30	48.0	36.4	—11.6		
Extra	Extra	26	46.6	38.6	—8.0		
Extra	Extra	27	45.6	38.3	—7.3		
		<i>m</i>	46.84	35.75	—11.09		
CALORIC GROUP G							
Basal	Basal	4	47.4	37.5	—9.9		
Basal	Basal	5	46.0	36.9	—9.1		
Basal	Extra	1	44.2	40.8	—3.4		
Basal	Extra	2	48.0	37.2	—10.8	36.9	45.0
Extra	Basal	11	46.6	38.6	—8.0		
Extra	Basal	12	39.8	31.3	—8.5	33.4	
Extra	Extra	8	45.0	28.6	—16.4		
Extra	Extra	9	44.3	32.5	—11.8		
		<i>m</i>	45.16	35.42	—9.74		
CALORIC GROUP T							
Basal	Basal	104	45.5	38.9	—6.6	42.9	44.0
Basal	Basal	105	47.8	31.3	—16.5	42.5	
Basal	Extra	101	47.3	40.6	—6.7	39.2	
Basal	Extra	102	50.3	39.9	—10.4		
Extra	Basal	111	48.6	39.4	—9.2	40.6	
Extra	Basal	112	48.4	37.2	—11.2	39.8	47.1
Extra	Extra	108	46.3	31.9	—14.4	40.9	
Extra	Extra	109	44.8	40.5	—4.3	39.9	46.8
		<i>m</i>	47.38	37.46	—9.92		
		<i>M</i>	46.47	36.57	—9.90		
		<i>SD</i>	2.03	3.74			

TABLE 463
HEMOGLOBIN, as gm. per 100 cc. of blood.

Supplement		Subject	Period					
Protein	Vitamin		C	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z								
Basal	Basal	122	15.0	12.3	—2.7	1.0	0.5	13.2
Basal	Basal	123	13.7	9.9	—3.8	0.5	1.2	
Basal	Extra	119	14.9	12.7	—2.2	0.6	0.2	
Basal	Extra	120	15.6	12.7	—2.9	0.8	1.4	
Extra	Basal	129	14.8	12.2	—2.7	1.0	1.0	15.7
Extra	Basal	130	14.8	10.9	—3.9	0.7	2.8	
Extra	Extra	126	14.6	11.1	—3.5	—0.2	0.2	15.3
Extra	Extra	127	16.6	11.6	—5.0	1.4	1.4	
		<i>m</i>	15.00	11.68	—3.34	0.72	1.09	
CALORIC GROUP L								
Basal	Basal	22	15.4	11.7	—3.7	0.8	2.4	15.4
Basal	Basal	23	16.8	12.9	—3.9	0.1	0.3	
Basal	Extra	19	15.6	12.4	—3.2	0.3	1.0	
Basal	Extra	20	15.0	10.6	—4.4	—0.2	1.2	
Extra	Basal	29	15.3	11.2	—4.1	1.6	2.2	15.1
Extra	Basal	30	15.0	12.5	—2.5	—0.1	0.2	
Extra	Extra	26	14.8	12.6	—2.2	—0.2	0.6	
Extra	Extra	27	14.4	11.2	—3.2	0.6	0.2	
		<i>m</i>	15.29	11.89	—3.40	0.36	1.01	
CALORIC GROUP G								
Basal	Basal	4	15.5	12.3	—3.2	—0.7	0.7	14.3
Basal	Basal	5	15.1	11.6	—3.5	0.0	0.5	
Basal	Extra	1	13.9	12.6	—1.3	—0.4	—0.9	14.5
Basal	Extra	2	16.0	11.1	—4.8	0.8	1.1	
Extra	Basal	11	14.5	11.6	—2.9	1.7	2.6	
Extra	Basal	12	13.3	10.1	—3.2	0.5	1.2	
Extra	Extra	8	15.0	9.0	—6.0	1.5	3.1	13.5
Extra	Extra	9	13.9	11.6	—2.3	1.3	1.4	
		<i>m</i>	14.65	11.24	—3.40	0.59	1.21	
CALORIC GROUP T								
Basal	Basal	104	14.6	12.3	—2.3	0.3	0.6	14.1
Basal	Basal	105	17.2	10.0	—7.2	2.4	3.7	14.8
Basal	Extra	101	15.7	11.8	—3.9	—0.3	1.6	
Basal	Extra	102	16.4	12.5	—3.9	—0.3	0.7	14.9
Extra	Basal	111	15.2	12.8	—2.4	0.5	0.5	
Extra	Basal	112	15.4	12.0	—3.4	0.7	0.0	
Extra	Extra	108	15.5	12.7	—2.8	—0.1	—0.1	
Extra	Extra	109	14.4	11.6	—2.8	0.0	0.6	
		<i>m</i>	15.55	11.96	—3.59	0.40	0.95	13.5
		<i>M</i>	15.12	11.70	—3.43	0.56	1.07	
		<i>SD</i>	0.88	1.01	1.17	0.68	1.02	

TABLE 464
TOTAL BLOOD VOLUME, in cc.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	5096	5255	159	3906	
Basal	Basal	123	5376	4842	—534		
Basal	Extra	119	4818	4232	—586	4208	
Basal	Extra	120	5595	5258	—337	4612	
Extra	Basal	129	6300	5548	—752		
Extra	Basal	130	6562	6500	—62	5537	6127
Extra	Extra	126	6152	5797	—355		
Extra	Extra	127	6397	5238	—1159		
		<i>m</i>	5787.0	5333.8	—453.2		
CALORIC GROUP L							
Basal	Basal	22	5725	4768	—957	4528	
Basal	Basal	23	5734	5239	—495		
Basal	Extra	19	5758	5125	—633		
Basal	Extra	20	5980	6072	92	4585	
Extra	Basal	29	6680	5006	—1674	6380	
Extra	Basal	30	5862	5044	—818		
Extra	Extra	26	5990	5654	—336		
Extra	Extra	27	6428	5944	—484		
		<i>m</i>	6019.6	5356.5	—663.1		
CALORIC GROUP G							
Basal	Basal	4	5138	4641	—497		
Basal	Basal	5	5400	5746	346		
Basal	Extra	1	6542	5993	—549		
Basal	Extra	2	6590	6768	178	7005	5787
Extra	Basal	11	5280	5876	596		
Extra	Basal	12	6190	5719	—471	6053	
Extra	Extra	8	5390	3506	—1884		
Extra	Extra	9	6248	5242	—1006		
		<i>m</i>	5847.3	5436.4	—410.9		
CALORIC GROUP T							
Basal	Basal	104	5651	5115	—536	4728	5379
Basal	Basal	105	5568	4997	—571	5016	
Basal	Extra	101	5156	5014	—142	4855	
Basal	Extra	102	6360	5595	—765		
Extra	Basal	111	5582	4319	—1263	4833	
Extra	Basal	112	5390	4514	—876	4829	5626
Extra	Extra	108	6702	6158	—544	4753	
Extra	Extra	109	5622	6619	997	5727	6284
		<i>m</i>	5753.9	5291.4	—462.5		
		<i>M</i>	5851.9	5354.5	—497.4		
		<i>SD</i>	526.4	717.6			

TABLE 465
RELATIVE BLOOD VOLUME, in cc. per kg. of body weight.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	77.4	110.0	32.6	76.9	
Basal	Basal	123	83.4	93.6	10.2		
Basal	Extra	119	72.8	84.4	11.6	80.1	
Basal	Extra	120	78.5	101.9	23.4	86.4	
Extra	Basal	129	95.5	104.3	8.8		
Extra	Basal	130	99.4	122.4	23.0	99.8	82.2
Extra	Extra	126	73.1	91.3	18.2		
Extra	Extra	127	99.3	107.3	8.0		
		<i>m</i>	84.92	101.90	16.98		
CALORIC GROUP L							
Basal	Basal	22	88.4	96.3	7.9	83.8	
Basal	Basal	23	82.7	102.0	19.3		
Basal	Extra	19	80.2	98.0	17.8		
Basal	Extra	20	93.0	127.8	34.8	83.4	
Extra	Basal	29	92.6	93.4	0.8	108.9	
Extra	Basal	30	85.9	95.7	9.8		
Extra	Extra	26	84.5	102.8	18.3		
Extra	Extra	27	84.7	107.0	22.3		
		<i>m</i>	86.50	102.88	16.38		
CALORIC GROUP G							
Basal	Basal	4	83.9	100.8	16.9		
Basal	Basal	5	66.1	98.7	32.6		
Basal	Extra	1	83.2	106.1	22.9		
Basal	Extra	2	89.4	118.1	28.7	114.6	77.2
Extra	Basal	11	80.7	118.8	38.1		
Extra	Basal	12	76.3	92.8	16.5	92.6	
Extra	Extra	8	82.2	71.5	—10.7		
Extra	Extra	9	85.6	91.2	5.6		
		<i>m</i>	80.92	99.75	18.82		
CALORIC GROUP T							
Basal	Basal	104	82.4	99.3	16.9	79.7	69.7
Basal	Basal	105	80.5	96.5	16.0	83.6	
Basal	Extra	101	79.9	104.4	24.5	84.7	
Basal	Extra	102	93.0	107.0	14.0		
Extra	Basal	111	88.4	87.6	—0.8	81.9	
Extra	Basal	112	86.9	86.2	—0.7	82.7	87.2
Extra	Extra	108	98.8	109.4	10.6	80.6	
Extra	Extra	109	70.8	112.3	41.5	88.9	80.8
		<i>m</i>	85.09	100.34	15.25		
		<i>M</i>	84.36	101.22	16.84		
		<i>SD</i>	8.18	11.68			

TABLE 466
TOTAL PLASMA VOLUME, in cc.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	2746	3206	460	2375	
Basal	Basal	123	3059	3404	345		
Basal	Extra	119	2599	2504	—95	2584	
Basal	Extra	120	2857	2960	103	2629	
Extra	Basal	129	3316	3393	77		
Extra	Basal	130	3540	4349	809	3472	3229
Extra	Extra	126	3393	3623	230		
Extra	Extra	127	3215	3232	17		
		<i>m</i>	3090.6	3333.9	243.3		
CALORIC GROUP L							
Basal	Basal	22	3087	3066	—21	2726	
Basal	Basal	23	3068	3238	170		
Basal	Extra	19	3018	3485	467		
Basal	Extra	20	3116	3995	879	2916	
Extra	Basal	29	3580	3374	—206	3713	
Extra	Basal	30	3046	3208	162		
Extra	Extra	26	3201	3471	270		
Extra	Extra	27	3496	3667	171		
		<i>m</i>	3201.5	3438.0	236.5		
CALORIC GROUP G							
Basal	Basal	4	2705	2969	264		
Basal	Basal	5	2913	3626	713		
Basal	Extra	1	3647	3548	—99		
Basal	Extra	2	3430	4250	820	4420	3183
Extra	Basal	11	2816	3608	792		
Extra	Basal	12	3727	3929	202	4031	
Extra	Extra	8	2968	2511	—457		
Extra	Extra	9	3478	3538	60		
		<i>m</i>	3210.5	3497.4	286.9		
CALORIC GROUP T							
Basal	Basal	104	3066	3120	54	2700	3012
Basal	Basal	105	2872	3432	560	2889	
Basal	Extra	101	2717	2978	261	2952	
Basal	Extra	102	3149	3363	214		
Extra	Basal	111	2866	2620	—246	2871	
Extra	Basal	112	2784	2835	51	2907	2976
Extra	Extra	108	3597	4193	596	2809	
Extra	Extra	109	3102	3938	836	3442	3343
		<i>m</i>	3019.1	3309.9	290.8		
		<i>M</i>	3130.4	3394.8	264.4		
		<i>SD</i>	310.7	469.8			

TABLE 467
RELATIVE PLASMA VOLUME, in cc. per kg. of body weight.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	41.7	67.0	25.3	46.8	
Basal	Basal	123	47.5	65.8	18.3		
Basal	Extra	119	39.3	49.9	10.6	49.2	
Basal	Extra	120	40.3	57.4	17.1	49.2	
Extra	Basal	129	50.3	63.7	13.4		
Extra	Basal	130	53.7	81.9	28.2	62.6	43.3
Extra	Extra	126	40.3	57.0	16.7		
Extra	Extra	127	49.9	66.3	16.4		
		<i>m</i>	45.38	63.62	18.25		
CALORIC GROUP L							
Basal	Basal	22	47.7	62.0	14.3	50.5	
Basal	Basal	23	44.3	63.0	18.7		
Basal	Extra	19	42.0	66.6	24.6		
Basal	Extra	20	48.4	84.1	35.7	53.0	
Extra	Basal	29	49.6	63.0	13.4	63.4	
Extra	Basal	30	44.6	60.9	16.3		
Extra	Extra	26	45.2	63.2	18.0		
Extra	Extra	27	45.9	66.0	20.1		
		<i>m</i>	45.96	66.10	20.14		
CALORIC GROUP G							
Basal	Basal	4	44.2	63.2	19.0		
Basal	Basal	5	35.7	62.2	26.6		
Basal	Extra	1	46.4	62.8	16.4		
Basal	Extra	2	46.5	74.2	27.7	72.3	42.4
Extra	Basal	11	43.0	72.9	29.9		
Extra	Basal	12	46.0	63.8	17.8	61.6	
Extra	Extra	8	45.2	51.1	5.9		
Extra	Extra	9	47.7	61.5	13.8		
		<i>m</i>	44.34	63.98	19.64		
CALORIC GROUP T							
Basal	Basal	104	44.8	60.7	15.9	45.5	39.0
Basal	Basal	105	41.6	66.3	24.7	48.2	
Basal	Extra	101	42.1	62.0	19.9	51.5	
Basal	Extra	102	46.0	64.3	18.3		
Extra	Basal	111	45.4	53.1	7.7	48.6	
Extra	Basal	112	44.8	54.1	9.3	49.8	46.1
Extra	Extra	108	53.1	74.5	21.4	47.6	
Extra	Extra	109	39.1	66.8	27.7	53.4	43.0
		<i>m</i>	44.61	62.72	18.11		
		<i>M</i>	45.07	64.11	19.04		
		<i>SD</i>	4.00	5.00			

TABLE 468
 BASAL OXYGEN CONSUMPTION, in cc. of oxygen (STP) used per minute.

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	235	146	124	—111	16	37	254
Basal	Basal	123	210	149	128	—82	20	48	
Basal	Extra	119	225	167	153	—72	—10	15	250
Basal	Extra	120	223	171	150	—73	—2	7	239
Extra	Basal	129	224	130	149	—75	—2	18	
Extra	Basal	130	219	148	126	—93	16	34	241
Extra	Extra	126	226	156	140	—86	—3	13	
Extra	Extra	127	242	149	136	—106	18	40	
		<i>m</i>	225.5	152.0	138.2	—87.2	6.6	26.5	
CALORIC GROUP L									
Basal	Basal	22	215	166	123	—92	8	48	241
Basal	Basal	23	240	166	162	—78	11	42	
Basal	Extra	19	210	143	122	—88	23	51	256
Basal	Extra	20	217	161	142	—75	13	41	
Extra	Basal	29	236	151	142	—94	7	37	256
Extra	Basal	30	214	144	123	—91	12	44	
Extra	Extra	26	242	145	141	—101	6	18	
Extra	Extra	27	229	172	145	—84	16	41	
		<i>m</i>	225.4	156.0	137.5	—87.9	12.0	40.2	
CALORIC GROUP G									
Basal	Basal	4	234	148	143	—91	15	62	220
Basal	Basal	5	236	189	155	—81	10	28	
Basal	Extra	1	239	167	150	—89	4	32	214
Basal	Extra	2	234	158	142	—92	21	49	
Extra	Basal	11	206	158	141	—65	19	51	215
Extra	Basal	12	262	180	148	—114	19	53	
Extra	Extra	8	210	132	118	—92	37	67	260
Extra	Extra	9	246	136	128	—118	37	67	
		<i>m</i>	233.4	158.5	140.6	—92.8	20.2	51.1	
CALORIC GROUP T									
Basal	Basal	104	218	141	128	—90	20	63	255
Basal	Basal	105	254	139	118	—136	45	85	
Basal	Extra	101	213	142	124	—89	34	72	214
Basal	Extra	102	228	162	145	—83	21	61	
Extra	Basal	111	228	155	131	—97	29	59	215
Extra	Basal	112	217	145	162	—55	1	35	
Extra	Extra	108	232	156	139	—93	28	54	260
Extra	Extra	109	235	186	175	—60	18	29	
		<i>m</i>	228.1	153.2	140.2	—87.9	24.5	57.2	
		<i>M</i>	228.1	154.9	139.2	—88.9	15.8	43.8	
		<i>SD</i>	13.5	14.8	14.0				

TABLE 469

BASAL OXYGEN CONSUMPTION, in cc. of oxygen (STP) used per minute per square meter of body surface (DuBois).

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	132	92	82	—50	10	20	135
Basal	Basal	123	114	87	76	—38	14	28	
Basal	Extra	119	120	100	95	—25	—6	6	133
Basal	Extra	120	121	102	94	—27	—2	2	123
Extra	Basal	129	118	74	87	—31	—2	6	
Extra	Basal	130	117	85	73	—44	10	19	129
Extra	Extra	126	107	82	77	—30	—2	6	
Extra	Extra	127	133	90	84	—49	10	20	
		<i>m</i>	120.2	89.0	83.5	—36.7	4.0	13.4	
CALORIC GROUP L									
Basal	Basal	22	119	98	76	—43	4	26	130
Basal	Basal	23	128	98	98	—30	7	22	
Basal	Extra	19	115	85	76	—39	13	29	130
Basal	Extra	20	121	97	90	—31	5	20	
Extra	Basal	29	126	89	84	—42	3	19	130
Extra	Basal	30	114	83	74	—40	6	21	
Extra	Extra	26	126	83	84	—42	2	5	
Extra	Extra	27	118	98	86	—32	9	22	
		<i>m</i>	120.9	91.4	83.5	—37.4	6.1	20.5	
CALORIC GROUP G									
Basal	Basal	4	135	92	92	—43	7	28	122
Basal	Basal	5	118	106	90	—28	5	12	
Basal	Extra	1	118	89	84	—34	2	14	
Basal	Extra	2	123	91	82	—41	14	25	
Extra	Basal	11	113	94	86	—27	11	25	
Extra	Basal	12	124	92	79	—45	9	24	
Extra	Extra	8	121	84	76	—45	21	35	
Extra	Extra	9	124	75	72	—52	19	32	
		<i>m</i>	122.0	90.4	82.6	—39.4	11.0	24.4	
CALORIC GROUP T									
Basal	Basal	104	118	83	77	—41	12	32	133
Basal	Basal	105	138	82	72	—66	26	44	117
Basal	Extra	101	116	85	78	—38	18	35	
Basal	Extra	102	116	95	88	—28	9	27	120
Extra	Basal	111	131	94	81	—50	15	27	
Extra	Basal	112	128	90	102	—26	—2	12	
Extra	Extra	108	128	92	85	—43	16	28	128
Extra	Extra	109	114	99	97	—17	9	11	
		<i>m</i>	123.6	90.0	85.0	—38.6	12.9	27.0	
		<i>M</i>	121.7	90.2	83.7	—38.0	8.5	21.3	
		<i>SD</i>	7.2	7.6	8.1	10.0	7.4	10.0	

TABLE 470
 ORAL TEMPERATURE, in degrees F., secured at the time the B.M.R.
 determinations were made.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12
CALORIC GROUP Z								
Basal	Basal	122	98.0	97.6	97.6	—0.4	—0.4	0.4
Basal	Basal	123	96.7	97.2	96.8	0.1	1.2	0.1
Basal	Extra	119	97.7	96.4	97.6	—0.1	0.0	0.0
Basal	Extra	120	97.0	96.0	97.0	0.0	—0.1	0.5
Extra	Basal	129	97.2	94.8	97.6	0.4	—0.8	—0.1
Extra	Basal	130	97.2	95.2	95.6	—1.6	0.8	1.4
Extra	Extra	126	96.1	95.6	97.0	0.9	—0.6	—0.8
Extra	Extra	127	97.2	94.2	97.0	—0.2	0.6	0.2
		<i>m</i>	97.13	95.88	97.02	—0.11	0.09	0.21
CALORIC GROUP L								
Basal	Basal	22	97.2	96.0	95.8	—1.4	0.9	1.2
Basal	Basal	23	97.2	96.8	97.3	0.1	—0.1	0.8
Basal	Extra	19	97.0	95.6	96.8	—0.2	0.6	0.8
Basal	Extra	20	97.0	95.5	96.2	—0.8	0.8	1.4
Extra	Basal	29	96.7	97.7	96.8	0.1	—0.1	0.5
Extra	Basal	30	96.2	95.4	96.6	0.4	0.0	0.6
Extra	Extra	26	97.6	96.8	97.6	0.0	—0.4	—0.4
Extra	Extra	27	96.8	97.2	96.6	—0.2	—0.6	0.5
		<i>m</i>	96.96	96.38	96.71	—0.25	0.14	0.68
CALORIC GROUP G								
Basal	Basal	4	97.7	94.8	96.9	—0.8	0.5	0.7
Basal	Basal	5	97.0	96.7	97.4	0.4	—0.4	—0.4
Basal	Extra	1	96.7	94.8	97.0	0.3	—0.3	0.4
Basal	Extra	2	97.6	94.8	97.3	—0.3	0.5	0.3
Extra	Basal	11	97.3	95.2	96.6	—0.7	—0.2	0.2
Extra	Basal	12	96.5	94.6	96.6	0.1	0.2	0.4
Extra	Extra	8	96.5	96.0	96.9	0.4	0.4	0.2
Extra	Extra	9	97.2	94.0	95.6	—1.6	1.5	1.8
		<i>m</i>	97.06	95.11	96.79	—0.27	0.28	0.45
CALORIC GROUP T								
Basal	Basal	104	96.9	95.1	96.4	—0.5	0.9	1.2
Basal	Basal	105	97.1	94.1	96.6	—0.5	0.9	1.4
Basal	Extra	101	97.4	95.6	97.2	—0.2	0.4	0.4
Basal	Extra	102	97.0	95.8	97.2	0.2	—0.8	0.2
Extra	Basal	111	97.8	97.8	97.6	—0.2	0.2	0.0
Extra	Basal	112	97.0	94.8	96.4	—0.6	0.0	0.4
Extra	Extra	108	97.4	96.6	96.6	—0.8	0.4	0.4
Extra	Extra	109	98.0	96.4	98.2	0.2	—0.2	0.1
		<i>m</i>	97.32	95.78	97.02	—0.30	0.22	0.51
		<i>M</i>	97.12	95.78	96.89	—0.23	0.18	0.46
		<i>SD</i>	0.47		0.60			

TABLE 471
URINARY NITROGEN EXCRETION, as gm. per 24 hours.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12
CALORIC GROUP Z								
Basal	Basal	122	15.0	9.2	7.6	—7.4	0.2	2.1
Basal	Basal	123	12.1	6.9	7.5	—4.6	0.7	3.0
Basal	Extra	119	13.1	8.6	8.3	—4.8	—2.9	1.1
Basal	Extra	120	13.9	7.3	7.9	—6.0	0.5	2.8
Extra	Basal	129	13.9	7.1	6.6	—7.3	3.9	10.8
Extra	Basal	130	13.0	9.1	5.5	—7.5	4.8	11.3
Extra	Extra	126	14.0	7.8	6.9	—7.1	3.2	10.7
Extra	Extra	127	12.0	8.2	7.2	—4.8	2.9	10.2
		<i>m</i>	13.38	8.02	7.19	—6.19	1.66	6.50
CALORIC GROUP L								
Basal	Basal	22	12.2	8.2	7.6	—4.6	0.7	3.9
Basal	Basal	23	13.7	8.6	8.0	—5.7	0.4	2.1
Basal	Extra	19	13.7	8.1	6.4	—7.3	0.9	4.0
Basal	Extra	20	14.4	9.2	7.9	—6.5	0.3	2.3
Extra	Basal	29	13.0	10.8	8.1	—4.9	2.2	9.5
Extra	Basal	30	13.1	7.3	7.0	—6.1	2.8	9.7
Extra	Extra	26	14.0	9.3	7.2	—6.8	2.9	9.9
Extra	Extra	27	14.9	7.9	8.1	—6.8	3.1	9.2
		<i>m</i>	13.63	8.68	7.54	—6.09	1.66	6.32
CALORIC GROUP G								
Basal	Basal	4	11.2	8.2	7.0	—4.2	1.2	4.0
Basal	Basal	5	12.2	6.1	8.0	—4.2	1.0	3.2
Basal	Extra	1	13.4	10.6	8.9	—4.5	0.9	3.7
Basal	Extra	2	10.4	6.9	6.4	—4.0	2.0	4.9
Extra	Basal	11	11.8	6.7	6.8	—5.0	4.0	11.3
Extra	Basal	12	13.8	8.9	8.4	—5.4	2.3	8.6
Extra	Extra	8	13.4	8.5	7.0	—6.4	2.5	12.4
Extra	Extra	9	14.9	7.4	8.8	—6.1	2.0	9.3
		<i>m</i>	12.63	7.91	7.66	—4.97	1.99	7.18
CALORIC GROUP T								
Basal	Basal	104	15.3	9.4	7.2	—8.1	1.7	5.0
Basal	Basal	105	13.4	10.4	7.2	—6.2	0.9	3.9
Basal	Extra	101	12.4	7.2	8.7	—3.7	—0.3	3.3
Basal	Extra	102	11.7	6.8	7.2	—4.5	1.3	3.2
Extra	Basal	111	13.5	8.8	7.7	—5.8	4.7	11.9
Extra	Basal	112	14.4	6.3	6.5	—7.9	5.3	9.9
Extra	Extra	108	10.6	6.9	5.8	—4.8	5.7	13.7
Extra	Extra	109	13.0	7.0	8.1	—4.9	4.8	10.5
		<i>m</i>	13.04	7.85	7.30	—5.74	3.01	7.68
		<i>M</i>	13.17	8.12	7.42	—5.75	2.08	6.92
		<i>SD</i>	1.24	1.2	0.83		1.9	3.9

TABLE 472

TOTAL PLASMA PROTEIN, corrected for non-protein nitrogen, as gm. per 100 cc

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	6.64	6.69	6.16	—0.48	0.49	0.80	6.08
Basal	Basal	123	6.58	6.00	5.81	—0.77	0.49	1.30	
Basal	Extra	119	6.37	6.71	6.41	0.04	0.00	0.74	
Basal	Extra	120	6.62	6.34	6.29	—0.33	—0.28	—0.26	6.38
Extra	Basal	129	6.49	6.36	6.05	—0.44	0.43	0.97	
Extra	Basal	130	6.33	6.12	6.13	—0.20	—0.40	—0.06	
Extra	Extra	126	6.67	6.25	5.52	—1.15	—0.22	—0.24	6.78
Extra	Extra	127	6.87	6.54	6.07	—0.80	0.56	0.92	
		<i>m</i>	6.571	6.376	6.055	—0.516	0.134	0.521	
CALORIC GROUP L									
Basal	Basal	22	6.68	6.43	5.92	—0.76	—0.07	0.51	6.34
Basal	Basal	23	6.81	6.59	6.48	—0.33	—0.02	—0.59	
Basal	Extra	19	6.72	6.61	5.44	—1.28	0.26	1.04	
Basal	Extra	20	6.32	5.91	5.25	—1.07	0.04	0.67	6.75
Extra	Basal	29	6.38	6.18	5.07	—1.31	0.17	0.61	
Extra	Basal	30	6.88	6.26	5.43	—1.45	0.57	0.67	
Extra	Extra	26	6.78	6.57	6.67	—0.11	0.58	0.46	6.75
Extra	Extra	27	6.41	6.42	5.87	—0.54	—0.27	—0.11	
		<i>m</i>	6.622	6.371	5.766	—0.856	0.158	0.408	
CALORIC GROUP G									
Basal	Basal	4	6.86	6.61	7.08	0.22	—0.94	—0.81	6.24
Basal	Basal	5	6.60	6.30	6.26	—0.34	—0.26	—0.23	6.71
Basal	Extra	1	6.02	5.75	5.80	—0.22	0.18	0.13	
Basal	Extra	2	7.01	6.66	6.51	—0.50	—0.16	—0.25	
Extra	Basal	11	6.68	6.87	6.38	—0.30	0.01	—0.03	6.71
Extra	Basal	12	6.40	5.68	5.30	—1.10	—0.06	0.31	
Extra	Extra	8	6.90	5.93	5.06	—1.84	0.54	1.52	
Extra	Extra	9	6.47	6.21	5.96	—0.51	—0.05	0.24	6.71
		<i>m</i>	6.618	6.251	6.044	—0.574	—0.092	0.110	
CALORIC GROUP T									
Basal	Basal	104	6.62	6.08	5.85	—0.77	0.59	1.45	7.04
Basal	Basal	105	7.27	6.90	6.03	—1.24	0.96	1.26	
Basal	Extra	101	7.54	6.17	5.89	—1.65	—0.19	0.62	
Basal	Extra	102	6.72	7.22	6.34	—0.38	—0.04	0.73	5.74
Extra	Basal	111	6.36	6.31	5.87	—0.49	0.03	0.28	
Extra	Basal	112	7.26	6.87	6.66	—0.60	0.58	0.40	
Extra	Extra	108	6.43	6.15	5.59	—0.84	0.67	1.47	6.24
Extra	Extra	109	6.67	6.72	6.36	—0.31	0.01	0.24	
		<i>m</i>	6.859	6.552	6.074	—0.785	0.326	0.806	
		<i>M</i>	6.668	6.388	5.985	—0.683	0.131	0.461	
		<i>SD</i>	0.312		0.485				

TABLE 473

PLASMA ALBUMIN, as gm. per 100 cc. (columns 1 to 4). RATIO OF PLASMA
ALBUMIN TO PLASMA GLOBULIN (columns 5 to 8).

			1	2	3	4	5	6	7	8
Supplement			Period							
Protein	Vitamin	Subject	C	S24	dS24	△R12	C	S24	dS24	△R12
CALORIC GROUP Z										
Basal	Basal	122	4.27	3.87	—0.40	0.77	1.80	1.69	—0.11	0.31
Basal	Basal	123	3.76	4.20	0.44	0.51	1.33	2.61	1.28	—0.65
Basal	Extra	119	4.94	4.40	—0.54	0.14	3.45	2.19	—1.26	—0.45
Basal	Extra	120	4.96	4.36	—0.60	—0.16	2.99	2.26	—0.73	0.04
Extra	Basal	129	4.18	3.97	—0.21	0.99	1.81	1.91	0.10	0.50
Extra	Basal	130	4.21	2.86	—1.35	0.98	1.99	0.87	—1.12	0.85
Extra	Extra	126	4.60	3.62	—0.98	0.20	2.22	1.91	—0.31	0.71
Extra	Extra	127	4.20	4.12	—0.08	0.02	1.57	2.11	0.54	—0.66
		<i>m</i>	4.390	3.925	—0.465	0.431	2.145	1.944	—0.201	0.081
CALORIC GROUP L										
Basal	Basal	22	4.52	3.85	—0.67	0.31	2.09	1.86	—0.23	—0.03
Basal	Basal	23	4.81	3.98	—0.83	0.00	2.40	1.59	—0.81	0.49
Basal	Extra	19	4.03	3.72	—0.31	0.62	1.50	2.16	0.66	—0.13
Basal	Extra	20	4.13	3.60	—0.53	0.33	1.89	2.18	0.29	—0.21
Extra	Basal	29	4.10	2.32	—1.78	1.70	1.80	0.84	—0.96	1.58
Extra	Basal	30	4.89	4.16	—0.73	0.78	2.46	3.28	0.82	0.58
Extra	Extra	26	3.91	4.12	0.21	0.58	1.36	1.62	0.26	0.31
Extra	Extra	27	4.17	3.98	—0.19	—0.22	1.86	2.11	0.25	—0.23
		<i>m</i>	4.320	3.716	—0.604	0.512	1.920	1.955	0.035	0.295
CALORIC GROUP G										
Basal	Basal	4	5.20	4.81	—0.39	—0.69	3.13	2.12	—1.01	—0.20
Basal	Basal	5	3.82	2.58	—1.24	1.38	1.37	0.70	—0.67	1.21
Basal	Extra	1	3.14	3.72	0.58	0.12	1.09	1.79	0.70	0.05
Basal	Extra	2	4.58	4.61	0.03	—0.57	1.88	2.43	0.55	—0.61
Extra	Basal	11	4.96	4.02	—0.94	—0.16	2.88	1.70	—1.18	—0.15
Extra	Basal	12	3.87	3.52	—0.35	0.40	1.53	1.98	0.45	0.34
Extra	Extra	8	3.78	3.44	—0.34	0.84	1.21	2.12	0.91	—0.26
Extra	Extra	9	4.40	4.40	0.00	—0.74	2.13	2.82	0.69	—1.38
		<i>m</i>	4.219	3.888	—0.331	0.072	1.902	1.957	0.055	—0.125
CALORIC GROUP T										
Basal	Basal	104	4.07	4.18	0.11	0.19	1.60	2.50	0.90	—1.01
Basal	Basal	105	4.54	4.08	—0.46	0.20	1.66	2.09	0.43	—0.67
Basal	Extra	101	5.17	4.16	—1.01	0.03	2.18	2.40	0.22	—0.59
Basal	Extra	102	3.68	3.86	0.18	0.20	1.21	1.56	0.35	—0.21
Extra	Basal	111	3.80	3.89	0.09	0.08	1.48	1.96	0.48	—0.14
Extra	Basal	112	3.95	3.16	—0.79	0.86	1.19	0.90	—0.29	0.42
Extra	Extra	108	4.19	3.90	—0.29	0.64	1.87	2.31	0.44	—0.51
Extra	Extra	109	4.02	4.12	0.10	—0.04	1.52	1.84	0.32	—0.22
		<i>m</i>	4.178	3.919	—0.259	0.270	1.589	1.945	0.356	—0.366
		<i>M</i>	4.277	3.862	—0.415	0.321	1.889	1.950	0.061	—0.029
		<i>SD</i>	0.485	0.538			0.591	0.563		

TABLE 474

PLASMA TOTAL CHOLESTEROL. Each sample contained heparin as an anticoagulant. All values are in mg. per 100 cc. of plasma.

Supplement			Period		
Protein	Vitamin	Subject	C	S24	R6
CALORIC GROUP Z					
Basal	Basal	122	201	186	218
Basal	Basal	123	200	159	
Basal	Extra	119	144	121	
Extra	Basal	129	181	185	
Extra	Basal	130	165	157	
Extra	Extra	126	155	161	
Extra	Extra	127	191	144	
CALORIC GROUP L					
Basal	Basal	23	140	127	171
Extra	Basal	29	188	139	
Extra	Basal	30	216	185	
Extra	Extra	26	164	157	
Extra	Extra	27	135	122	
CALORIC GROUP G					
Basal	Basal	4	198	142	
Basal	Basal	5	135	135	
Basal	Extra	1	157	168	
Extra	Basal	12	144	147	
Extra	Extra	8	157	130	
Extra	Extra	9	139	128	
CALORIC GROUP T					
Basal	Basal	105	178	140	202
Basal	Extra	101	181	171	
Extra	Basal	111	192	146	
Extra	Basal	112	171	188	
Extra	Extra	109	162	128	
		M	169.3	150.7	191
		SD	24.0	21.7	193

TABLE 475

URINARY CREATININE EXCRETION, as gm. per 24 hours (columns 1 to 4). URINARY RIBOFLAVIN EXCRETION, as micrograms for 24 hours (columns 5 to 8).

Supplement		Subject	1	2	3	4	5	6	7	8
Protein	Vitamin		Period							
			S12	S24	Δ R6	Δ R12	S12	S24	R6	R12
CALORIC GROUP Z										
Basal	Basal	122	1.26	0.96	0.01	0.34	45	117	75	175
Basal	Basal	123	0.95	0.96	-0.04	0.36	46	66	119	315
Basal	Extra	119	1.34	1.18	-0.25	0.05	36	58	1150	1425
Basal	Extra	120	1.08	0.82	0.28	0.55	56	64	1113	1307
Extra	Basal	129	0.97	0.82	0.02	0.31	16	30	68	165
Extra	Basal	130	1.13	0.89	0.07	0.37	83	62	104	216
Extra	Extra	126	1.32	1.14	-0.07	0.26	59	38	1372	1513
Extra	Extra	127	1.05	0.87	-0.04	0.27	66	97	1036	1096
		<i>m</i>	1.138	0.955	-0.002	0.314	50.9	66.5	629.6	776.5
CALORIC GROUP L										
Basal	Basal	22	1.07	0.71	0.27	0.55	89	46	82	123
Basal	Basal	23	1.28	1.01	0.13	0.40	75	43	126	190
Basal	Extra	19	1.05	1.01	-0.05	0.36	67	90	1149	1194
Basal	Extra	20	0.99	0.77	0.19	0.46	59	33	1171	799
Extra	Basal	29	0.98	0.92	0.09	0.62	90	70	63	178
Extra	Basal	30	1.37	0.93	0.04	0.47	75	30	140	217
Extra	Extra	26	1.51	0.77	0.33	0.61	44	34	1248	1432
Extra	Extra	27	0.90	0.98	-0.13	0.32	25	25	1224	1310
		<i>m</i>	1.144	0.888	0.109	0.474	65.5	46.4	650.4	680.4
CALORIC GROUP G										
Basal	Basal	4	1.15	0.83	0.21	0.60	88	79	205	247
Basal	Basal	5	1.65	1.23	-0.09	0.27	40	24	153	236
Basal	Extra	1	1.65	1.24	-0.07	0.48	160	132	1283	1239
Basal	Extra	2	1.20	0.89	0.06	0.29	41	58	1370	981
Extra	Basal	11	0.77	1.00	0.05	0.46	12	26	129	193
Extra	Basal	12	1.30	1.04	0.10	0.79	26	110	66	90
Extra	Extra	8	1.85	0.84	0.19	0.79	16	33	942	1370
Extra	Extra	9	1.10	1.20	-0.15	0.24	139	109	1139	1290
		<i>m</i>	1.333	1.034	0.038	0.490	65.2	71.4	660.9	705.8
CALORIC GROUP T										
Basal	Basal	104	1.42	0.90	0.18	0.68	102	54	121	196
Basal	Basal	105	1.27	0.84	0.11	0.30	20	36	84	254
Basal	Extra	101	0.67	0.97	0.01	0.44	54	27	1103	1109
Basal	Extra	102	1.24	0.92	0.09	0.46	54	176	1101	1345
Extra	Basal	111	1.08	0.83	0.29	0.73	42	76	221	266
Extra	Basal	112	1.22	1.57	-0.53	-0.05	24	35	63	186
Extra	Extra	108	0.93	1.36	-0.32	0.20	26	46	1074	1291
Extra	Extra	109	1.40	1.19	-0.05	0.33	27	26	1266	1447
		<i>m</i>	1.154	1.072	-0.028	0.386	43.6	59.5	629.1	761.8
		<i>M</i>	1.192	0.987	0.029	0.416	56.3	60.9	642.5	731.1
		<i>SD</i>	0.255	0.191			34.7	36.8	544.4	553.5

TABLE 476

URINARY THIAMINE, in micrograms (columns 1 to 4). URINARY PYRAMIN, in micrograms of 2-methyl-4-amino-5-ethoxymethylpyrimidine hydrochloride (columns 5 to 8). All values are expressed on a 24-hour basis.

			1	2	3	4	5	6	7	8
Supplement			Period							
Protein	Vitamin	Subject	S12	S24	△R6	△R12	S12	S24	△R6	△R12
CALORIC GROUP Z										
Basal	Basal	122	148	168	76	258	122	138	30	101
Basal	Basal	123	59	109	99	319	119	121	55	120
Basal	Extra	119	119	127	475	732	161	143	105	179
Basal	Extra	120	77	100	501	580	134	150	108	148
Extra	Basal	129	136	127	55	223	120	124	48	151
Extra	Basal	130	123	57	72	191	158	109	51	129
Extra	Extra	126	42	41	658	962	120	100	148	195
Extra	Extra	127	177	200	405	316	134	122	110	103
		<i>m</i>	110.1	116.1	292.6	447.6	133.5	125.9	81.9	140.8
CALORIC GROUP L										
Basal	Basal	22	177	171	185	404	158	141	85	151
Basal	Basal	23	273	305	48	133	168	188	20	80
Basal	Extra	19	54	67	883	991	120	114	143	214
Basal	Extra	20	156	300	622	832	111	149	83	153
Extra	Basal	29	244	258	9	77	131	153	41	80
Extra	Basal	30	128	55	161	325	148	94	98	146
Extra	Extra	26	119	117	570	755	165	107	158	210
Extra	Extra	27	88	98	632	683	149	152	90	106
		<i>m</i>	154.9	171.4	388.8	525.0	143.8	137.2	89.8	142.5
CALORIC GROUP G										
Basal	Basal	4	224	217	204	329	143	134	80	154
Basal	Basal	5	85	108	214	341	124	144	76	81
Basal	Extra	1	83	191	694	811	123	200	133	195
Basal	Extra	2	73	125	907	1055	114	124	134	235
Extra	Basal	11	141	169	99	161	146	146	56	84
Extra	Basal	12	106	193	138	121	140	137	69	94
Extra	Extra	8	89	73	734	885	121	108	144	255
Extra	Extra	9	166	126	778	819	113	130	136	175
		<i>m</i>	120.9	150.2	471.0	565.2	128.0	140.4	103.5	159.1
CALORIC GROUP T										
Basal	Basal	104	65	144	203	369	114	142	106	178
Basal	Basal	105	84	102	150	308	126	134	72	193
Basal	Extra	101	68	102	756	901	110	158	160	260
Basal	Extra	102	172	126	1127	1066	154	137	162	241
Extra	Basal	111	168	135	290	235	160	146	79	130
Extra	Basal	112	216	63	360	442	150	83	112	115
Extra	Extra	108	99	41	792	909	112	104	202	198
Extra	Extra	109	86	191	529	605	138	169	189	221
		<i>m</i>	119.8	113.0	525.9	604.4	133.0	134.1	135.2	192.0
		<i>M</i>	126.4	137.7	419.6	535.6	134.6	134.4	102.6	158.6
		<i>SD</i>	58.4	68.6	311.5	315.7	18.2	25.4	46.3	54.2

TABLE 477

SERUM BILIRUBIN, as Ehrlich units per 100 cc. The one-minute values (column 1) represent the bilirubin determined directly. The total values (column 2) represent the above bilirubin plus that liberated by the addition of methyl alcohol.

URINARY UROBILINOGEN, as Ehrlich units per 24 hours (columns 3 and 4).

Samples were collected from 1 to 3 P.M.

			1	2	3	4
Supplement			Period			
Protein	Vitamin	Subject	S20	S20	S20	S21
CALORIC GROUP Z						
Basal	Basal	122	0.13	0.52	0.79	
Basal	Basal	123	0.08	0.54	2.14	1.43
Basal	Extra	119	0.12	0.77	1.26	
Basal	Extra	120	0.06	0.44	1.27	
Extra	Basal	129	0.13	0.92	1.74	2.07
Extra	Basal	130	0.09	0.59	1.58	1.23
Extra	Extra	126	0.15	0.93	0.73	
Extra	Extra	127	0.09	0.70	1.03	
CALORIC GROUP L						
Basal	Basal	22	0.05	1.17	1.81	1.74
Basal	Basal	23	0.18	0.95	2.87	1.51
Basal	Extra	19	0.08	0.73	2.06	1.01
Basal	Extra	20	0.16	1.02	2.86	1.41
Extra	Basal	29	0.12	0.67	5.29	1.81
Extra	Basal	30	0.07	0.47	2.22	1.75
Extra	Extra	26	0.12	0.46	0.54	
Extra	Extra	27	0.09	0.80	0.88	
CALORIC GROUP G						
Basal	Basal	4	0.13	0.64	0.64	
Basal	Basal	5	0.06	0.51		
Basal	Extra	1	0.01	0.46	1.71	1.47
Basal	Extra	2	0.11	0.93	1.60	2.57
Extra	Basal	11	0.07	0.59	1.49	
Extra	Basal	12	0.12	0.82	1.46	
Extra	Extra	8	0.09	0.40	1.48	
Extra	Extra	9	0.10	0.51	1.56	1.29
CALORIC GROUP T						
Basal	Basal	104	0.07	0.61	1.37	1.38
Basal	Basal	105	0.07	0.97	0.97	
Basal	Extra	101	0.12	0.93	1.26	
Basal	Extra	102	0.16	0.74	1.50	1.30
Extra	Basal	111	0.05	0.74	1.18	
Extra	Basal	112	0.00	0.36	0.95	
Extra	Extra	108	0.11	0.54	4.11	1.22
Extra	Extra	109	0.16	0.83	2.21	2.77
		<i>M</i>	0.098	0.696	1.695	
		<i>SD</i>	0.042	0.209	0.999	

TABLE 478

URINARY VOLUME, in cc. per 24 hours. The data for Subjects 2, 29, 108, and 129 at S24 were not included since their fluid intake was restricted at that time.

Supplement		Subject	Period				
Protein	Vitamin		C	S12	S24	R6	R12
CALORIC GROUP Z							
Basal	Basal	122	1825	1793	4470	3187	1973
Basal	Basal	123	975	1797	2417	4190	2143
Basal	Extra	119	1070	923		2075	1890
Basal	Extra	120	1885	2240	3877	2923	2853
Extra	Basal	129	1230	1177	1223	1333	1483
Extra	Basal	130	1230	1130	1305	1480	1530
Extra	Extra	126	1082	1760	2857	1750	2107
Extra	Extra	127	1375	2190	2043	1423	1557
		<i>m</i>	1334.0	1626.2	2598.9	2295.1	1942.0
CALORIC GROUP L							
Basal	Basal	22	1330	2807	2847	3087	1887
Basal	Basal	23	1415	2333	2393	2477	2563
Basal	Extra	19	1120	1400	2640	2520	2073
Basal	Extra	20	1360	1297	1817	2137	1737
Extra	Basal	29	1595	2708		3015	2403
Extra	Basal	30	1225	1587	1850	1973	2437
Extra	Extra	26	920	2845	3543	4933	2070
Extra	Extra	27	1237	917	1087	1530	1860
		<i>m</i>	1275.2	1986.8	2311.0	2709.0	2128.8
CALORIC GROUP G							
Basal	Basal	4	1055	1530	1607	1847	1957
Basal	Basal	5	1150	1090	2183	1403	1580
Basal	Extra	1	915	2295	3363	3290	3193
Basal	Extra	2	938	663		3057	1277
Extra	Basal	11	1080	1283	1447	1503	1427
Extra	Basal	12	1120	940	2523	3400	2907
Extra	Extra	8	1760	2840	2450	2560	3027
Extra	Extra	9	1260	2523	2637	1930	2180
		<i>m</i>	1159.8	1645.5	2315.7	2373.8	2193.4
CALORIC GROUP T							
Basal	Basal	104	1505	2705	3543	3673	2973
Basal	Basal	105	1090	2010	2197	1680	1460
Basal	Extra	101	1315	1260	1623	2223	2113
Basal	Extra	102	1498	1910	1903	2597	2047
Extra	Basal	111	1355	1497	1997	2420	2182
Extra	Basal	112	1205	1217	900	1690	2170
Extra	Extra	108	1145	2440		2887	2360
Extra	Extra	109	1148	1170	1250	1990	1987
		<i>m</i>	1282.6	1776.1	1916.1	2395.0	2161.5
		<i>M</i>	1262.9	1758.7	2285.4	2443.2	2106.4
		<i>SD</i>	249.3	681.5	891.7	870.5	497.8

TABLE 479

BLOOD SUGAR, in mg. per 100 cc. Venous blood obtained at the 27th minute of a 30-minute walk on a motor-driven treadmill at 3.5 miles per hour and a 10 per cent grade (columns 1 to 5). BASAL BLOOD SUGAR, in mg. per 100 cc. Samples obtained in the morning before the subjects got out of bed (columns 6 to 8).

			1	2	3	4	5	6	7	8
Supplement			Period							
Protein	Vitamin	Subject	C	S24	dS24	△R6	△R12	S24	△R6	△R12
CALORIC GROUP Z										
Basal	Basal	122	77	60	—17	7	—3	70	—8	7
Basal	Basal	123	77	59	—18	1	—7	73	—8	0
Basal	Extra	119	69	57	—12	2	—6	63	6	5
Basal	Extra	120	81	68	—13	0	—10	66	2	4
Extra	Basal	129	74	56	—18	3	6	62	3	20
Extra	Basal	130	76	58	—18	6	—8	61	2	15
Extra	Extra	126	73	61	—12	—3	—8	62	0	11
Extra	Extra	127	64	54	—10	8	11	63	1	2
		<i>m</i>	73.9	59.1	—14.8	3.0	—3.1	65.0	0	8.0
CALORIC GROUP L										
Basal	Basal	22	75	58	—17	2	—2	62	2	16
Basal	Basal	23	70	56	—14	11	2	70	—6	—1
Basal	Extra	19	66	54	—12	13	2	66	—9	12
Basal	Extra	20	70	58	—12	4	—3	64	2	9
Extra	Basal	29	72	49	—23	9	1	62	—1	11
Extra	Basal	30	63	59	—4	8	—12	56	6	7
Extra	Extra	26	69	56	—13	10	1	58	8	10
Extra	Extra	27	79	63	—16	4	0	70	3	6
		<i>m</i>	70.5	56.6	—13.9	7.6	—1.4	63.5	5	8.8
CALORIC GROUP G										
Basal	Basal	4	79	70	—9	—4	0	72	—6	2
Basal	Basal	5	68	52	—16	12	4	64	4	12
Basal	Extra	1	73	55	—18	6	2	62	0	12
Basal	Extra	2	73	66	—7	—2	—8	61	4	15
Extra	Basal	11	69	59	—10	6	3	70	—4	8
Extra	Basal	12	76	70	—6	—3	—12	64	7	14
Extra	Extra	8	71	50	—21	8	3	64	8	8
Extra	Extra	9	59	56	—3	8	—3	60	7	18
		<i>m</i>	71.0	59.8	—11.2	3.9	—1.4	64.6	2	11.1
CALORIC GROUP T										
Basal	Basal	104	69	63	—6	0	—11	62	1	3
Basal	Basal	105	66	60	—6	—3	3	60	—1	8
Basal	Extra	101	84	55	—29	2	6	59	9	12
Basal	Extra	102	72	54	—18	3	0	56	2	19
Extra	Basal	111	73	55	—18	10	7	62	7	10
Extra	Basal	112	67	52	—15	7	10	62	6	15
Extra	Extra	108	63	57	—6	4	1	60	9	8
Extra	Extra	109	69	56	—13	3	4	66	3	5
		<i>m</i>	70.4	56.5	—13.9	3.5	2.5	60.9	4	10.0
		<i>M</i>	71.4	58.0	—13.4	4.5	—0.8	63.5		9.5
		<i>SD</i>	5.6	5.6				4.3		

TABLE 480
RESPIRATION, as respiratory cycles per minute, from basal metabolism charts.

Supplement			Period						
Protein	Vitamin	Subject	C	S12	S24	dS24	ΔR6	ΔR12	R20
CALORIC GROUP Z									
Basal	Basal	122	11.1	10.0	8.8	—2.3	0.2	1.0	17.5
Basal	Basal	123	13.8	13.6	13.6	—0.2	—0.2	1.4	
Basal	Extra	119	11.4	8.6	10.0	—1.4	—3.0	—1.9	
Basal	Extra	120	7.2	6.7	6.0	—1.2	0.2	1.1	11.5
Extra	Basal	129	13.8	8.4	10.1	—3.7	—0.2	0.8	
Extra	Basal	130	12.8	10.9	6.9	—5.9	3.0	3.2	
Extra	Extra	126	11.9	8.7	6.9	—5.0	0.5	1.2	10.0
Extra	Extra	127	8.2	9.8	8.8	0.6	—0.6	—0.3	
		<i>m</i>	11.28	9.59	8.89	—2.39	—0.01	0.81	
CALORIC GROUP L									
Basal	Basal	22	5.7	5.5	6.1	0.4	0.0	0.1	10.3
Basal	Basal	23	7.5	6.5	6.5	—1.0	—0.3	0.6	
Basal	Extra	19	11.6	10.2	9.8	—1.8	0.6	—0.8	
Basal	Extra	20	12.8	10.4	11.8	—1.0	—0.7	—1.2	13.3
Extra	Basal	29	13.0	11.5	10.5	—2.5	1.3	—0.2	
Extra	Basal	30	10.5	8.8	10.0	—0.5	1.9	—1.5	
Extra	Extra	26	7.8	10.8	9.9	2.1	1.0	1.1	13.3
Extra	Extra	27	13.4	11.8	12.1	—1.3	—1.9	—1.2	
		<i>m</i>	10.29	9.44	9.59	—0.70	0.24	—0.39	
CALORIC GROUP G									
Basal	Basal	4	9.2	8.7	10.1	0.9	—0.6	—0.3	10.8
Basal	Basal	5	12.9	12.4	13.1	0.2	—0.1	0.7	
Basal	Extra	1	11.0	10.5	10.0	—1.0	—1.5	0.4	
Basal	Extra	2	11.0	8.0	5.1	—5.9	0.9	3.1	
Extra	Basal	11	10.6	9.4	9.6	—1.0	1.4	—0.7	
Extra	Basal	12	13.9	12.4	10.9	—3.0	0.9	2.2	
Extra	Extra	8	10.1	9.2	11.5	1.4	0.7	—0.5	
Extra	Extra	9	14.5	9.6	10.8	—3.7	—0.7	1.3	
		<i>m</i>	11.65	10.02	10.14	—1.51	0.12	0.78	
CALORIC GROUP T									
Basal	Basal	104	11.9	9.5	10.0	—1.9	1.5	1.5	12.4
Basal	Basal	105	16.2	13.5	12.0	—4.2	2.5	2.2	
Basal	Extra	101	12.4	9.5	8.5	—3.9	1.9	4.6	
Basal	Extra	102	15.3	11.5	11.2	—4.1	—0.4	0.8	7.6
Extra	Basal	111	11.1	9.8	11.5	0.4	0.3	0.5	
Extra	Basal	112	10.1	10.2	11.0	0.9	1.8	1.3	
Extra	Extra	108	11.0	9.2	9.8	—1.2	—0.4	0.3	13.0
Extra	Extra	109	12.8	11.0	12.6	—0.2	1.0	0.4	
		<i>m</i>	12.60	10.52	10.82	—1.78	1.02	1.45	
		<i>M</i>	11.45	9.89	9.86	—1.59	0.35	0.66	13.9
		<i>SD</i>	2.41		2.13				

TABLE 481

VENTILATION, in liters per minute, calculated from basal metabolism charts.

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	4.9	3.3	2.7	—2.2	0.2	0.6	
Basal	Basal	123	5.5	4.5	4.0	—1.5	—0.2	0.8	6.0
Basal	Extra	119	4.6	3.3	3.2	—1.4	—0.2	0.2	4.3
Basal	Extra	120	4.1	3.2	2.8	—1.3	0.0	0.5	
Extra	Basal	129	5.0	3.0	3.2	—1.8	0.5	0.4	8.6
Extra	Basal	130	4.6	3.5	2.8	—1.8	0.4	0.9	
Extra	Extra	126	4.8	3.4	2.7	—2.1	0.0	0.8	
Extra	Extra	127	4.4	3.0	2.8	—1.6	0.7	0.9	5.0
		<i>m</i>	4.74	3.40	3.03	—1.71	0.18	0.64	
CALORIC GROUP L									
Basal	Basal	22	4.7	2.6	2.8	—1.9	0.5	0.8	
Basal	Basal	23	4.0	3.2	2.8	—1.2	0.7	0.8	4.1
Basal	Extra	19	4.4	2.8	2.8	—1.6	1.0	1.0	
Basal	Extra	20	5.5	4.1	4.1	—1.4	0.0	0.4	
Extra	Basal	29	5.2	4.1	3.6	—1.6	0.5	0.4	
Extra	Basal	30	4.2	3.1	2.8	—1.4	0.8	0.6	
Extra	Extra	26	4.5	3.7	2.9	—1.6	0.4	0.7	4.8
Extra	Extra	27	4.7	3.7	3.4	—1.3	—0.2	0.3	
		<i>m</i>	4.65	3.42	3.15	—1.50	0.46	0.62	
CALORIC GROUP G									
Basal	Basal	4	4.1	3.6	3.4	—0.7	—0.2	0.3	5.1
Basal	Basal	5	5.2	4.4	4.3	—0.9	—0.6	—0.1	
Basal	Extra	1	5.3	3.8	4.0	—1.3	—0.1	0.5	
Basal	Extra	2	4.1	3.5	2.6	—1.5	0.4	1.0	
Extra	Basal	11	4.1	3.1	3.2	—0.9	0.5	0.3	
Extra	Basal	12	6.4	4.9	4.9	—1.5	—0.7	0.3	
Extra	Extra	8	4.6	2.8	2.9	—1.7	0.9	1.1	
Extra	Extra	9	5.3	3.0	3.1	—2.2	0.7	1.3	
		<i>m</i>	4.89	3.64	3.55	—1.34	0.11	0.59	
CALORIC GROUP T									
Basal	Basal	104	4.6	3.2	3.0	—1.6	1.0	1.3	5.1
Basal	Basal	105	5.1	4.0	4.5	—0.6	0.4	0.4	
Basal	Extra	101	4.9	3.7	3.9	—1.0	0.2	0.4	4.0
Basal	Extra	102	6.0	3.0	3.3	—2.7	0.3	1.0	
Extra	Basal	111	4.8	3.4	3.8	—1.0	0.0	0.7	
Extra	Basal	112	4.8	2.9	3.2	—1.6	0.8	0.7	3.9
Extra	Extra	108	4.6	3.6	3.4	—1.2	0.3	0.7	
Extra	Extra	109	5.3	4.3	4.3	—1.0	0.6	1.0	5.5
		<i>m</i>	5.01	3.51	3.67	—1.34	0.45	0.78	
		<i>M</i>	4.82	3.49	3.35	—1.47	0.30	0.66	
		<i>SD</i>	0.56		0.61				

TABLE 482

RESPIRATORY EFFICIENCY, as cc. of oxygen removed per liter of ventilation, calculated from basal metabolism charts.

Supplement			Period						
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	47.7	43.3	45.4	—2.3	1.9	3.3	
Basal	Basal	123	39.2	33.6	30.8	—8.4	7.8	7.1	42.2
Basal	Extra	119	48.0	50.4	48.1	0.1	1.0	1.5	57.8
Basal	Extra	120	54.4	54.0	53.5	—0.9	0.8	—5.4	
Extra	Basal	129	44.8	42.9	46.5	1.7	—7.0	—1.4	27.6
Extra	Basal	130	47.6	42.4	44.7	—2.9	0.2	—1.0	
Extra	Extra	126	46.1	46.7	51.1	5.0	—0.5	—7.5	
Extra	Extra	127	55.0	49.0	52.5	—2.5	—7.7	—4.2	48.7
		<i>m</i>	47.85	45.29	46.58	—1.27	—0.44	—0.95	
CALORIC GROUP L									
Basal	Basal	22	45.7	62.9	42.0	—3.7	—0.9	4.2	
Basal	Basal	23	59.5	52.3	56.4	—3.1	—6.9	—0.2	58.2
Basal	Extra	19	48.6	50.8	44.9	—3.7	—6.6	1.6	
Basal	Extra	20	40.9	39.0	35.0	—5.9	4.2	5.1	
Extra	Basal	29	45.8	37.6	40.0	—5.8	—3.4	4.1	
Extra	Basal	30	51.2	47.8	43.3	—7.9	—4.7	4.3	
Extra	Extra	26	52.5	39.1	49.4	—3.1	—4.3	—4.7	53.2
Extra	Extra	27	48.9	47.5	42.9	—6.0	7.7	6.8	
		<i>m</i>	49.14	47.12	44.24	—4.90	—1.86	2.65	
CALORIC GROUP G									
Basal	Basal	4	57.4	42.4	41.6	—15.8	8.8	12.6	43.0
Basal	Basal	5	45.7	43.9	36.3	—9.4	7.5	7.7	
Basal	Extra	1	45.2	44.6	38.2	—7.0	1.8	2.9	
Basal	Extra	2	57.2	45.5	54.2	—3.0	—0.5	—3.2	
Extra	Basal	11	49.4	52.2	45.6	—3.8	—1.6	9.4	
Extra	Basal	12	42.1	36.1	30.7	—11.4	12.2	8.2	
Extra	Extra	8	47.4	45.3	40.2	—7.2	1.2	6.0	
Extra	Extra	9	46.6	45.1	40.8	—5.8	3.3	3.3	
		<i>m</i>	48.88	44.39	40.95	—7.93	4.09	5.86	
CALORIC GROUP T									
Basal	Basal	104	47.4	44.2	43.0	—4.4	—6.7	1.5	50.8
Basal	Basal	105	48.6	35.0	25.1	—23.5	9.1	16.0	
Basal	Extra	101	44.2	36.8	31.9	—12.3	6.4	14.0	54.2
Basal	Extra	102	38.0	53.2	43.6	5.6	2.7	3.7	
Extra	Basal	111	46.8	45.7	35.8	—11.0	6.7	6.6	
Extra	Basal	112	46.4	49.8	50.2	3.8	—8.3	0.7	55.3
Extra	Extra	108	49.4	44.3	40.8	—8.6	3.6	6.2	
Extra	Extra	109	46.0	43.1	40.2	—5.8	—0.9	—2.1	47.6
		<i>m</i>	45.85	44.01	38.82	—7.03	1.58	5.82	
		<i>M</i>	47.93	45.20	42.65	—5.28	0.84	3.35	
		<i>SD</i>	4.94		7.38				

TABLE 483
VITAL CAPACITY, taken in seated position, in liters.

Supplement		Subject	Period				
Protein	Vitamin		C	S12	S24	dS24	Δ R12
CALORIC GROUP Z							
Basal	Basal	122	4.9	4.9	4.8	—0.1	0.0
Basal	Basal	123	5.4	5.4	5.1	—0.3	0.1
Basal	Extra	119	4.8	4.7	4.6	—0.2	0.2
Basal	Extra	120	4.4	3.8	4.2	—0.2	0.0
Extra	Basal	129	5.0	4.4	4.3	—0.7	0.5
Extra	Basal	130	5.4	5.3	5.2	—0.2	0.0
Extra	Extra	126	5.5	5.5	5.2	—0.3	0.0
Extra	Extra	127	4.9	4.6	4.4	—0.5	0.1
		<i>m</i>	5.04	4.82	4.73	—0.31	0.11
CALORIC GROUP L							
Basal	Basal	22	6.0	6.0	5.8	—0.2	0.0
Basal	Basal	23	5.5	5.2	5.1	—0.4	0.1
Basal	Extra	19	4.8	4.4	4.0	—0.8	0.5
Basal	Extra	20	5.1	4.8	4.1	—1.0	0.6
Extra	Basal	29	5.4	4.6	4.7	—0.7	0.3
Extra	Basal	30	4.6	4.3	3.8	—0.8	0.2
Extra	Extra	26	5.3	5.1	5.0	—0.3	0.0
Extra	Extra	27	5.1	5.1	4.7	—0.4	0.3
		<i>m</i>	5.22	4.93	4.65	—0.57	0.25
CALORIC GROUP G							
Basal	Basal	4	3.6	3.5	3.8	0.2	0.1
Basal	Basal	5	5.2	4.9	4.7	—0.5	0.4
Basal	Extra	1	5.8	5.6	5.3	—0.5	0.2
Basal	Extra	2	5.0	4.7	4.7	—0.3	0.1
Extra	Basal	11	5.1	4.9	4.6	—0.5	0.5
Extra	Basal	12	5.5	5.3	5.3	—0.2	0.1
Extra	Extra	8	4.9	4.6	4.4	—0.5	0.5
Extra	Extra	9	6.2	5.7	5.7	—0.5	0.3
		<i>m</i>	5.16	4.90	4.81	—0.35	0.28
CALORIC GROUP T							
Basal	Basal	104	4.9	4.4	4.2	—0.7	0.4
Basal	Basal	105	5.1	5.0	4.9	—0.2	0.3
Basal	Extra	101	5.3	5.0	4.5	—0.8	0.5
Basal	Extra	102	5.3	5.4	5.3	0.0	0.0
Extra	Basal	111	6.3	6.2	6.2	0.1	0.0
Extra	Basal	112	4.2	3.9	3.7	0.5	0.3
Extra	Extra	108	5.6	5.7	5.6	0.0	0.1
Extra	Extra	109	5.3	5.1	4.9	0.4	0.3
		<i>m</i>	5.25	5.09	4.91	—0.34	0.24
		<i>M</i>	5.17	4.94	4.78	—0.39	0.22
		<i>SD</i>	0.54	0.62	0.61		

TABLE 484

PULSE RATE, in beats per minute, taken at the time of determination of the basal metabolic rate.

Supplement			Period						
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	56	38	37	—19	1	8	
Basal	Basal	123	53	43	37	—16	3	9	66
Basal	Extra	119	55	44	45	—10	—1	12	58
Basal	Extra	120	55	41	42	—13	0	6	
Extra	Basal	129	47	30	34	—13	3	9	59
Extra	Basal	130	52	28	27	—25	6	17	
Extra	Extra	126	44	30	31	—13	2	5	
Extra	Extra	127	59	32	38	—21	3	12	66
		<i>m</i>	52.6	35.8	36.4	—16.2	2.1	9.8	
CALORIC GROUP L									
Basal	Basal	22	58	32	36	—22	2	14	
Basal	Basal	23	69	44	47	—22	5	17	64
Basal	Extra	19	52	35	38	—14	6	14	
Basal	Extra	20	59	34	41	—18	4	13	
Extra	Basal	29	55	42	42	—13	2	6	
Extra	Basal	30	60	36	37	—23	5	15	
Extra	Extra	26	52	30	30	—22	8	15	56
Extra	Extra	27	46	32	33	—13	5	11	
		<i>m</i>	56.4	35.6	38.0	—18.4	4.6	13.1	
CALORIC GROUP G									
Basal	Basal	4	62	31	38	—24	5	22	52
Basal	Basal	5	60	43	48	—12	—6	1	
Basal	Extra	1	47	28	29	—18	5	9	
Basal	Extra	2	61	35	33	—28	13	22	60
Extra	Basal	11	53	34	41	—12	3	11	
Extra	Basal	12	50	33	34	—16	2	7	
Extra	Extra	8	49	33	38	—11	5	8	
Extra	Extra	9	48	31	34	—14	4	11	
		<i>m</i>	53.8	33.5	36.9	—16.9	3.9	11.2	
CALORIC GROUP T									
Basal	Basal	104	49	34	34	—15	6	11	51
Basal	Basal	105	72	33	33	—39	10	18	
Basal	Extra	101	58	36	34	—24	10	18	50
Basal	Extra	102	63	42	45	—18	5	19	
Extra	Basal	111	62	42	39	—23	7	13	
Extra	Basal	112	53	30	41	—12	2	13	50
Extra	Extra	108	52	38	36	—16	8	15	
Extra	Extra	109	55	36	42	—13	2	14	56
		<i>m</i>	58.0	36.4	38.0	—20.0	6.2	13.1	
		<i>M</i>	55.2	35.3	37.3	—17.9	4.2	12.3	
		<i>SD</i>	6.5	5.0	5.2				

TABLE 485

OXYGEN CONSUMPTION PER HEART BEAT. The basal oxygen consumption in cc. per minute was divided by the pulse rate obtained at the same time.

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	4.2	3.8	3.4	—0.8	0.3	0.2	3.8
Basal	Basal	123	4.0	3.5	3.5	—0.5	0.2	0.3	
Basal	Extra	119	4.1	3.8	3.4	—0.7	—0.2	—0.5	
Basal	Extra	120	4.1	4.2	3.6	—0.5	—0.1	—0.3	4.1
Extra	Basal	129	4.8	4.3	4.4	—0.4	—0.4	—0.5	
Extra	Basal	130	4.2	5.3	4.7	0.5	—0.4	—1.1	
Extra	Extra	126	5.1	5.2	4.5	—0.6	—0.3	—0.3	3.7
Extra	Extra	127	4.1	4.7	3.6	—0.5	0.2	—0.1	
		<i>m</i>	4.32	4.35	3.89	—0.44	—0.09	—0.29	
CALORIC GROUP L									
Basal	Basal	22	3.7	5.2	3.4	—0.3	0.0	0.0	3.8
Basal	Basal	23	3.5	3.8	3.4	—0.1	—0.1	—0.2	
Basal	Extra	19	4.0	4.1	3.2	—0.8	0.1	0.1	
Basal	Extra	20	3.7	4.7	3.5	—0.2	—0.1	—0.1	4.6
Extra	Basal	29	4.3	3.6	3.4	—0.9	0.0	0.3	
Extra	Basal	30	3.6	4.0	3.3	—0.3	—0.1	—0.1	
Extra	Extra	26	4.7	4.8	4.7	0.0	—0.8	—1.2	
Extra	Extra	27	5.0	5.4	4.4	—0.6	—0.2	—0.2	
		<i>m</i>	4.06	4.45	3.66	—0.40	—0.15	—0.18	
CALORIC GROUP G									
Basal	Basal	4	3.8	4.8	3.8	0.0	—0.1	—0.4	4.2
Basal	Basal	5	3.9	4.4	3.2	— .7	0.7	0.5	3.7
Basal	Extra	1	5.1	6.0	5.2	0.1	—0.7	—0.4	
Basal	Extra	2	3.8	4.5	4.3	0.5	—0.8	—0.8	
Extra	Basal	11	3.9	4.6	3.4	—0.5	0.2	0.3	
Extra	Basal	12	5.2	5.5	4.4	—0.8	0.2	0.5	
Extra	Extra	8	4.3	4.0	3.1	—1.2	0.5	0.9	
Extra	Extra	9	5.1	4.4	3.8	—1.3	0.5	0.5	0.14
		<i>m</i>	4.39	4.78	3.90	—0.49	0.06		
CALORIC GROUP T									
Basal	Basal	104	4.4	4.1	3.8	—0.6	—0.1	0.4	5.0
Basal	Basal	105	3.5	4.2	3.6	0.1	0.2	0.4	4.3
Basal	Extra	101	3.7	3.9	3.6	—0.1	0.0	0.2	
Basal	Extra	102	3.6	3.9	3.2	—0.4	0.1	0.0	
Extra	Basal	111	3.7	3.7	3.4	—0.3	0.1	0.3	4.3
Extra	Basal	112	4.1	4.8	4.0	—0.1	—0.2	—0.4	
Extra	Extra	108	4.5	4.1	3.9	—0.6	—0.1	—0.1	
Extra	Extra	109	4.3	5.2	4.2	—0.1	0.2	—0.6	4.6
		<i>m</i>	3.98	4.24	3.71	—0.26	0.02	0.02	
		<i>M</i>	4.19	4.45	3.79	—0.40	—0.04	0.07	
		<i>SD</i>	5.13		.535	0.42	0.34	0.48	

TABLE 486
SYSTOLIC BLOOD PRESSURE, in mm. of mercury, during supine rest.

Supplement			Period						
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	111	107	102	—9	4	—6	
Basal	Basal	123	96	86	82	—14	10	8	94
Basal	Extra	119	109	98	92	—17	2	2	106
Basal	Extra	120	119	100	96	—23	—4	4	
Extra	Basal	129	98	92	94	—4	—4	8	112
Extra	Basal	130	98	92	92	—6	2	0	
Extra	Extra	126	107	100	94	—13	2	—2	
Extra	Extra	127	116	114	97	—19	13	11	114
		<i>m</i>	106.7	98.6	93.6	—13.1	3.1	3.1	
CALORIC GROUP L									
Basal	Basal	22	121	108	96	—25	6	8	
Basal	Basal	23	109	90	92	—17	10	14	106
Basal	Extra	19	106	96	100	—6	—5	—5	
Basal	Extra	20	101	92	92	—9	4	2	
Extra	Basal	29	106	102	98	—8	2	—2	
Extra	Basal	30	112	99	90	—22	16	6	
Extra	Extra	26	101	88	94	—7	6	0	104
Extra	Extra	27	111	110	104	—7	—8	2	
		<i>m</i>	108.4	98.1	95.8	—12.6	3.9	3.1	
CALORIC GROUP G									
Basal	Basal	4	104	100	88	—16	6	8	96
Basal	Basal	5	98	96	84	—14	8	23	
Basal	Extra	1	113	118	110	—3	4	8	
Basal	Extra	2	109	101	104	—5	—8	—4	96
Extra	Basal	11	101	100	92	—9	1	2	
Extra	Basal	12	115	100	104	—11	2	—2	
Extra	Extra	8	96	96	90	—6	16	0	
Extra	Extra	9	104	98	85	—19	5	9	
		<i>m</i>	105.0	101.1	94.6	—10.4	4.2	5.5	
CALORIC GROUP T									
Basal	Basal	104	100	94	86	—14	8	20	100
Basal	Basal	105	115	92	94	—21	12	8	
Basal	Extra	101	108	94	94	—14	2	8	104
Basal	Extra	102	104	96	86	—18	12	16	
Extra	Basal	111	107	88	90	—17	8	14	
Extra	Basal	112	109	110	100	—9	0	14	110
Extra	Extra	108	99	114	118	19	—14	—22	
Extra	Extra	109	106	103	90	—16	8	15	106
		<i>m</i>	106.0	98.9	94.8	—11.2	4.5	9.1	
		<i>M</i>	106.5	99.2	94.7	—11.8	3.9	5.2	
		<i>SD</i>	6.7		7.7				

TABLE 487

PULSE PRESSURE, in mm. of mercury, in supine rest. This is the difference between systolic and diastolic blood pressure.

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	36	32	32	—4	2	—4	30
Basal	Basal	123	35	32	22	—13	12	8	
Basal	Extra	119	39	34	28	—11	0	—10	
Basal	Extra	120	47	38	32	—15	4	2	
Extra	Basal	129	30	38	40	10	—6	—2	36
Extra	Basal	130	32	34	34	2	2	—6	
Extra	Extra	126	40	30	34	—6	—4	—8	36
Extra	Extra	127	38	30	25	—13	7	9	
		<i>m</i>	37.1	33.5	30.9	—6.2	2.1	—1.4	
CALORIC GROUP L									
Basal	Basal	22	49	38	38	—11	2	2	46
Basal	Basal	23	41	30	34	—7	4	6	
Basal	Extra	19	39	20	28	—11	—9	—1	
Basal	Extra	20	37	34	28	—9	4	4	
Extra	Basal	29	30	20	22	—8	6	—2	34
Extra	Basal	30	41	29	30	—11	—2	2	
Extra	Extra	26	29	26	28	—1	—4	—2	
Extra	Extra	27	40	32	38	—2	—12	—2	
		<i>m</i>	38.2	28.6	30.7	—7.5	—1.4	0.9	
CALORIC GROUP G									
Basal	Basal	4	36	32	28	—8	—2	2	32
Basal	Basal	5	32	34	26	—6	2	5	
Basal	Extra	1	47	38	30	—17	8	14	33
Basal	Extra	2	37	38	30	—7	0	0	
Extra	Basal	11	33	36	34	1	—7	—4	
Extra	Basal	12	45	32	32	—13	0	4	
Extra	Extra	8	28	26	30	2	6	—4	34
Extra	Extra	9	34	18	21	—13	11	9	
		<i>m</i>	36.5	31.8	28.9	—7.6	2.2	3.2	
CALORIC GROUP T									
Basal	Basal	104	30	34	30	0	4	10	36
Basal	Basal	105	36	24	26	—10	6	2	
Basal	Extra	101	34	20	20	—14	10	10	36
Basal	Extra	102	30	32	30	0	0	4	
Extra	Basal	111	38	28	30	—8	2	4	30
Extra	Basal	112	37	40	42	5	—18	—8	
Extra	Extra	108	37	30	38	1	—4	—8	34
Extra	Extra	109	35	32	26	—9	8	11	
		<i>m</i>	34.6	30.0	30.2	—4.4	1.0	3.1	
		<i>M</i>	36.6	31.0	30.2	—6.4	1.0	1.5	
		<i>SD</i>	5.4	5.8	5.4	6.5	6.7	6.3	

TABLE 488

VENOUS BLOOD PRESSURE, in supine position, measured at the antecubital fossa as cm. of saline solution above heart level (columns 1 to 4). POSTURAL ADJUSTMENT (tilt table test) (columns 5 to 8); scored according to Crampton (1906).

			1	2	3	4	5	6	7	8
Supplement			Period							
Protein	Vitamin	Subject	S24	△R6	△R12	R20	C	S24	dS24	R20
CALORIC GROUP Z										
Basal	Basal	122	4.2	1.1	0.2		45	10	—35	
Basal	Basal	123	8.4	—1.2	0.0	10.6	55	50	—5	65
Basal	Extra	119	4.4	0.9	2.5	19.9	65	60	—5	50
Basal	Extra	120	2.1	1.1	2.1		70	60	—10	
Extra	Basal	129	4.1	1.1	0.4	7.8	35	20	—15	40
Extra	Basal	130	9.6	—1.8	—3.3		60	60	0	
Extra	Extra	126	3.7	4.1	3.8		55	40	—15	
Extra	Extra	127	3.4	1.7	1.8	9.2	35	50	15	45
		<i>m</i>	4.99	0.88	0.88		52.5	43.8	—8.8	
CALORIC GROUP L										
Basal	Basal	22	4.4	0.3	2.3		25	30	5	
Basal	Basal	23	4.1	0.3	2.6	9.6	30	—5	—35	65
Basal	Extra	19	7.1	—1.3	—2.4		40	75	35	
Basal	Extra	20	4.7	—0.8	1.5		70	40	—30	
Extra	Basal	29	4.2	2.5	2.2		45	55	10	
Extra	Basal	30	4.1	—0.2	1.7		40	30	—10	
Extra	Extra	26	2.7	2.2	4.0	12.3	50	35	—15	50
Extra	Extra	27	4.2	1.1	2.6					
		<i>m</i>	4.44	0.51	1.81		42.9	37.1	—5.7	
CALORIC GROUP G										
Basal	Basal	4	3.2	0.7	1.0	12.8	60	15	—45	65
Basal	Basal	5	5.6	—1.3	0.4		55	45	—10	
Basal	Extra	1	4.0	1.1	3.8		35	65	30	
Basal	Extra	2	6.8	—1.8	—2.3	8.8	50	25	—25	60
Extra	Basal	11	4.6	—0.1	1.0		75	60	—15	
Extra	Basal	12	4.3	1.7	2.8		25	25	0	
Extra	Extra	8	6.7	0.2	—0.2		15	35	20	
Extra	Extra	9	4.6	1.4	1.5		25	45	20	
		<i>m</i>	4.98	0.24	1.00		42.5	39.4	—3.1	
CALORIC GROUP T										
Basal	Basal	104	6.3	2.9	2.1	8.6	40	50	10	55
Basal	Basal	105	3.5	—0.9	4.0		60	45	—15	
Basal	Extra	101	4.3	1.6	2.9	10.2	15	15	0	40
Basal	Extra	102	5.6	0.1	0.1		25	25	0	
Extra	Basal	111	2.8	2.0	3.9		40	25	—15	
Extra	Basal	112	4.5	0.5	4.6	12.0	75	80	5	75
Extra	Extra	108	6.7	—1.9	—1.5		35	—5	—40	
Extra	Extra	109	4.8	—0.2	0.9	10.2	45	65	20	55
		<i>m</i>	4.81	0.51	2.12		41.9	37.5	—4.4	
		<i>M</i>	4.80	0.534	1.47		45.0	39.5	—5.5	
		<i>SD</i>	1.64	1.46	1.98		16.9	21.6		

TABLE 489

ELECTROCARDIOGRAM IN THE CONTROL PERIOD. K_{QT} (column 1), K_{syst} (column 2), P amplitude in lead 2 as mv. $\times 10$ (column 3), ΣQRS for the three limb leads as mv. $\times 10$ (column 4), T amplitude in lead 2 as mv. $\times 10$ (column 5), QRS axis in degrees (column 6), T axis in degrees (column 7).

Supplement		Subject	1	2	3	4	5	6	7
Protein	Vitamin								
CALORIC GROUP Z									
Basal	Basal	122	358	339	0.6	30.1	2.4	77	38
Basal	Basal	123	365	333	1.1	22.5	4.0	78	56
Basal	Extra	119	376	331	0.4	19.2	1.7	70	46
Basal	Extra	120	362	308	1.0	22.1	0.3	64	43
Extra	Basal	129	348	311	0.7	19.8	3.7	70	71
Extra	Basal	130	400	364	0.8	21.6	1.7	57	60
Extra	Extra	126	380	349	1.3	28.4	2.8	97	5
Extra	Extra	127	382	369	1.0	11.5	2.1	78	74
		<i>m</i>	371.4	338.0	0.86	21.90	2.34	73.9	38.4
CALORIC GROUP L									
Basal	Basal	22	405	322	1.1	21.5	2.3	87	59
Basal	Basal	23	378	334	1.1	33.4	3.8	66	70
Basal	Extra	19	359	318	1.0	25.7	3.3	68	38
Basal	Extra	20	335	345	0.5	7.3	1.7	77	64
Extra	Basal	29	390	348	0.9	9.2	0.9	50	64
Extra	Basal	30	364	353	1.6	30.8	3.3	81	63
Extra	Extra	26	364	317	1.5	25.3	3.7	67	51
Extra	Extra	27	376	321	0.4	11.6	2.0	38	33
		<i>m</i>	371.4	332.2	1.01	20.60	2.62	66.8	55.2
CALORIC GROUP G									
Basal	Basal	4	365	323	1.8	24.7	2.7	104	49
Basal	Basal	5	390	344	1.5	21.9	2.7	68	66
Basal	Extra	1	386	323	0.8	12.9	1.4	37	21
Basal	Extra	2	356	325	1.4	30.7	3.1	65	51
Extra	Basal	11	383	355	1.1	31.3	4.1	77	58
Extra	Basal	12	365	318	1.4	24.8	3.1	62	38
Extra	Extra	8	397	343	1.3	30.6	3.6	54	52
Extra	Extra	9	353	326	0.0	25.7	3.7	83	62
		<i>m</i>	374.4	332.1	1.16	25.32	3.05	68.8	49.6
CALORIC GROUP T									
Basal	Basal	104	374	355	1.1	27.9	2.5	67	41
Basal	Basal	105	372	335	0.8	19.0	1.1	30	6
Basal	Extra	101	373	332	0.9	34.3	3.0	68	61
Basal	Extra	102	390	356	1.3	21.0	2.4	70	56
Extra	Basal	111	350	350	1.3	31.5	3.7	76	60
Extra	Basal	112	346	318	0.7	39.6	4.8	78	49
Extra	Extra	108	362	342	0.6	19.5	1.7	51	43
Extra	Extra	109	340	330	1.4	16.4	4.0	75	59
		<i>m</i>	363.4	339.8	1.01	26.15	2.90	64.4	46.9
		<i>M</i>	370.1	335.5	1.01	23.49	2.73	68.4	47.5
		<i>SD</i>	17.5	15.9	0.40	7.75	1.07	15.9	23.5

TABLE 490
ELECTROCARDIOGRAM, SUM OF THE T AMPLITUDES, for the three standard leads,
in mv. $\times 10$.

Supplement		Subject	Period					
Protein	Vitamin		C	S12	S24	dS24	ΔR12	R32
CALORIC GROUP Z								
Basal	Basal	122	5.1	4.8	3.4	—1.7	2.8	5.0
Basal	Basal	123	8.3	4.0	1.9	—6.4	3.1	8.2
Basal	Extra	119	3.5	7.4	4.4	0.9	0.9	6.3
Basal	Extra	120	1.0	5.0	3.6	2.6	2.0	
Extra	Basal	129	6.8	6.8	4.8	—2.0	0.8	9.8
Extra	Basal	130	3.5	6.7	3.4	—0.1	1.1	8.2
Extra	Extra	126	6.5	4.9	3.2	—3.3	0.8	
Extra	Extra	127	3.4	6.7	4.7	1.3	1.3	6.8
		<i>m</i>	4.76	5.79	3.67	—1.09	1.60	
CALORIC GROUP L								
Basal	Basal	22	4.6	2.7	1.6	—3.0	1.9	2.8
Basal	Basal	23	7.2	7.3	5.8	—1.4	3.2	
Basal	Extra	19	6.2	3.5	1.2	—5.0	2.4	6.5
Basal	Extra	20	3.5	4.0	2.6	—0.9	3.0	
Extra	Basal	29	1.8	3.2	2.4	0.6	1.9	4.7
Extra	Basal	30	6.6	2.7	2.9	—3.7	1.2	7.4
Extra	Extra	26	7.6	5.1	4.4	—3.2	3.7	7.6
Extra	Extra	27	4.4	5.9	3.3	—1.1	1.5	8.0
		<i>m</i>	5.24	4.30	3.03	—2.21	2.35	
CALORIC GROUP G								
Basal	Basal	4	5.8	6.5	2.5	—3.3	3.3	5.1
Basal	Basal	5	5.4	6.6	4.2	—1.2	2.6	7.2
Basal	Extra	1	3.5	1.8	1.4	—2.1	0.8	4.4
Basal	Extra	2	6.4	7.5	2.6	—3.8	3.5	8.8
Extra	Basal	11	8.4	13.1	8.2	—0.2	2.9	9.2
Extra	Basal	12	7.0	4.4	0.8	—6.2	3.1	
Extra	Extra	8	7.4	4.8	3.9	—3.5	4.1	
Extra	Extra	9	7.3	1.7	2.5	—4.8	5.9	6.4
		<i>m</i>	6.40	5.80	3.26	—3.14	3.28	
CALORIC GROUP T								
Basal	Basal	104	5.1	4.8	3.1	—2.0	2.0	
Basal	Basal	105	3.3	5.7	2.5	—0.8	2.2	
Basal	Extra	101	5.7	8.3	3.4	—2.3	4.6	
Basal	Extra	102	4.9	7.4	3.5	—1.4	1.1	
Extra	Basal	111	7.5	3.7	3.7	—3.8	1.8	
Extra	Basal	112	9.5	8.6	9.5	0.0	—0.5	7.9
Extra	Extra	108	3.5	2.5	1.0	—2.5	2.9	
Extra	Extra	109	8.1	11.2	6.5	—1.6	1.6	7.5
		<i>m</i>	5.95	6.52	4.15	—1.80	1.96	
		<i>M</i>	5.59	5.60	3.53	—2.06	2.30	
		<i>SD</i>	2.51	2.55	1.92	2.84	1.31	

TABLE 491
ELECTROCARDIOGRAM, R AMPLITUDE IN LEAD 1, in mv. $\times 10$.

Supplement			Period					
Protein	Vitamin	Subject	C	S12	S24	dS24	△R12	R32
CALORIC GROUP Z								
Basal	Basal	122	5.0	2.0	2.1	—2.9	0.3	6.0
Basal	Basal	123	2.4	0.5	0.2	—2.2	0.0	4.9
Basal	Extra	119	2.7	3.4	1.6	—1.1	1.9	4.4
Basal	Extra	120	4.7	3.1	2.4	—2.3	0.8	
Extra	Basal	129	3.7	3.1	2.7	—1.0	0.5	8.7
Extra	Basal	130	3.1	2.4	1.5	—1.6	0.7	6.5
Extra	Extra	126	1.0	0.3	0.3	—0.7	0.1	
Extra	Extra	127	1.6	1.3	0.7	—0.9	0.8	4.3
		<i>m</i>	3.03	2.01	1.44	—1.59	0.64	
CALORIC GROUP L								
Basal	Basal	22	1.5	0.8	0.4	—1.1	0.6	2.0
Basal	Basal	23	6.2	3.8	2.9	—3.3	1.3	
Basal	Extra	19	3.8	1.2	1.1	—2.7	0.1	11.1
Basal	Extra	20	0.9	1.7	0.7	—0.2	1.2	
Extra	Basal	29	2.8	1.4	1.2	—1.6	1.1	7.1
Extra	Basal	30	2.5	1.1	0.5	—2.0	0.8	3.9
Extra	Extra	26	5.0	2.1	1.5	—3.5	1.0	8.2
Extra	Extra	27	2.5	1.7	1.2	—1.3	0.2	
		<i>m</i>	3.15	1.72	1.19	—1.96	0.79	5.8
CALORIC GROUP G								
Basal	Basal	4	1.2	0.9	0.9	—0.3	0.1	2.2
Basal	Basal	5	5.0	2.9	2.2	—2.8	—0.5	6.3
Basal	Extra	1	2.8	1.2	0.7	—2.1	1.0	4.9
Basal	Extra	2	7.1	3.8	3.6	—3.5	1.7	9.2
Extra	Basal	11	3.7	2.1	1.8	—1.9	0.9	4.4
Extra	Basal	12	6.4	2.6	2.1	—4.3	1.2	
Extra	Extra	8	8.8	3.7	3.0	—5.8	2.3	
Extra	Extra	9	1.6	0.7	0.6	—1.0	0.1	2.4
		<i>m</i>	4.57	2.24	1.86	—2.71	0.85	
CALORIC GROUP T								
Basal	Basal	104	5.2	2.5	1.1	—4.1	2.6	
Basal	Basal	105	4.2	1.7	1.3	—2.9	1.3	
Basal	Extra	101	6.0	3.6	1.6	—4.4	2.8	
Basal	Extra	102	3.5	1.9	1.4	—2.1	1.7	
Extra	Basal	111	4.1	1.5	0.7	—3.4	2.0	
Extra	Basal	112	4.4	3.0	2.0	—2.4	1.2	5.8
Extra	Extra	108	4.4	3.6	1.3	—3.1	1.1	
Extra	Extra	109	2.3	1.6	1.1	—1.2	0.7	3.3
		<i>m</i>	4.26	2.42	1.31	—2.75	1.68	
		<i>M</i>	3.75	2.10	1.45	—2.30	0.99	
		<i>SD</i>	1.89	1.05	0.85	1.31	0.78	

TABLE 492
ELECTROCARDIOGRAM, R AMPLITUDE IN LEAD 2, in mv. $\times 10$.

Supplement			Period					
Protein	Vitamin	Subject	C	S12	S24	dS24	ΔR12	R32
CALORIC GROUP Z								
Basal	Basal	122	12.7	10.2	9.6	—3.1	—1.3	9.5
Basal	Basal	123	9.4	6.3	4.7	—4.7	0.3	7.4
Basal	Extra	119	8.5	10.3	8.6	0.1	—0.6	11.2
Basal	Extra	120	10.2	13.5	10.4	0.2	—0.2	
Extra	Basal	129	9.9	7.6	6.2	—3.7	—1.5	8.6
Extra	Basal	130	6.4	4.8	3.7	—2.7	0.5	6.7
Extra	Extra	126	11.7	9.2	6.7	—5.0	1.1	
Extra	Extra	127	4.1	5.3	3.9	—0.2	0.5	6.0
		<i>m</i>	9.11	8.40	6.72	—2.39	—0.15	
CALORIC GROUP L								
Basal	Basal	22	7.6	6.3	4.7	—2.9	0.8	5.0
Basal	Basal	23	16.7	11.7	8.3	—8.4	2.8	
Basal	Extra	19	10.6	8.9	6.0	—4.6	1.5	8.8
Basal	Extra	20	2.9	5.8	4.3	1.4	1.2	
Extra	Basal	29	3.8	5.6	4.3	0.5	1.2	7.8
Extra	Basal	30	13.9	7.5	5.8	—8.1	1.5	11.2
Extra	Extra	26	12.3	7.1	5.2	—7.1	0.7	12.7
Extra	Extra	27	2.4	4.8	4.1	1.7	—0.6	3.3
		<i>m</i>	8.78	7.21	5.34	—3.44	1.14	
CALORIC GROUP G								
Basal	Basal	4	8.2	7.2	4.3	—3.9	3.3	8.9
Basal	Basal	5	8.3	8.7	7.3	—1.0	—0.4	10.0
Basal	Extra	1	3.9	4.1	3.3	—0.6	—0.3	5.6
Basal	Extra	2	14.4	9.3	5.9	—8.5	1.5	9.6
Extra	Basal	11	13.6	9.3	8.0	—5.6	1.5	10.2
Extra	Basal	12	11.1	7.4	6.0	—5.1	0.4	
Extra	Extra	8	14.0	9.4	7.3	—6.7	1.4	
Extra	Extra	9	12.2	10.6	7.4	—4.8	1.8	8.8
		<i>m</i>	10.71	8.25	6.19	—4.52	1.15	
CALORIC GROUP T								
Basal	Basal	104	10.0	8.5	6.7	—3.3	0.9	
Basal	Basal	105	5.8	6.6	4.3	—1.5	1.2	
Basal	Extra	101	14.9	9.4	7.1	—7.8	0.8	
Basal	Extra	102	10.4	7.9	5.4	—5.0	1.5	
Extra	Basal	111	15.5	9.0	7.3	—8.2	2.2	
Extra	Basal	112	18.0	12.4	13.1	—4.9	2.0	16.0
Extra	Extra	108	8.1	5.1	3.9	—4.2	1.0	
Extra	Extra	109	5.7	7.3	5.6	—0.1	0.7	6.5
		<i>m</i>	11.05	8.28	6.68	—4.37	1.29	
		<i>M</i>	9.91	8.03	6.23	—3.68	0.86	
		<i>SD</i>	4.15		2.18			

TABLE 493

ELECTROCARDIOGRAM, DURATION OF MECHANICAL SYSTOLE, in seconds \times 100.
Measured as the interval between first and second heart sounds.

Supplement		Subject	Period					R32
Protein	Vitamin		C	S12	S24	dS24	△R12	
CALORIC GROUP Z								
Basal	Basal	122	35	38	38	3	—2	33
Basal	Basal	123	31	38	40	9	—2	
Basal	Extra	119	30	37	38	8	0	
Basal	Extra	120	28	35	35	7	1	
Extra	Basal	129	33	39	36	3	1	
Extra	Basal	130	30	42	43	13	—3	
Extra	Extra	126	34	41	41	7	2	
Extra	Extra	127	30	39	39	9	—1	
		<i>m</i>	31.4	38.6	38.8	7.4	—0.5	
CALORIC GROUP L								
Basal	Basal	22	31	40	39	8	—3	33
Basal	Basal	23	31	38	36	5	—2	
Basal	Extra	19	31	39	39	8	—3	
Basal	Extra	20	35	39	37	2	—3	
Extra	Basal	29	33	40	39	6	—1	
Extra	Basal	30	32	38	40	8	—2	
Extra	Extra	26	34	42	40	6	—2	
Extra	Extra	27	35	38	42	7	—4	
		<i>m</i>	32.8	39.2	39.0	6.2	—2.5	
CALORIC GROUP G								
Basal	Basal	4	31	39	35	4	0	32
Basal	Basal	5	30	35	38	8	—2	
Basal	Extra	1	33	45	45	12	—5	
Basal	Extra	2	32	38	43	11	—3	
Extra	Basal	11	39	37	39	0	—4	
Extra	Basal	12	34	40	39	5	—2	
Extra	Extra	8	32	38	39	7	—2	
Extra	Extra	9	36	42	43	7	—4	
		<i>m</i>	33.4	39.2	40.1	6.7	—2.8	
CALORIC GROUP T								
Basal	Basal	104	36	44	42	6	—3	34
Basal	Basal	105	27	43	40	13	—3	
Basal	Extra	101	32	40	39	7	—3	
Basal	Extra	102	31	38	38	7	—4	
Extra	Basal	111	33	38	40	7	—3	
Extra	Basal	112	34	39	38	4	—2	
Extra	Extra	108	34	42	41	7	—2	
Extra	Extra	109	32	36	36	4	—1	
		<i>m</i>	32.4	40.0	39.3	6.9	—2.6	
		<i>M</i>	32.5	39.3	39.3	6.8	—2.1	
		<i>SD</i>	2.5	2.4	2.4	2.9	1.6	

TABLE 494

RECOVERY OF THE PULSE RATE after aerobic work (columns 1 to 5). RATIO OF OXYGEN CONSUMPTION TO PULSE RATE (oxygen pulse) for the last 5 minutes of a 30-minute walk on a motor-driven treadmill at 3.5 miles per hour and 10 per cent grade (columns 6 to 9).

Supplement			1	2	3	4	5	6	7	8	9
			Period								
Protein	Vitamin	Subject	C	S24	dS24	ΔR6	R20	C	S24	dS24	R20
CALORIC GROUP Z											
Basal	Basal	122	106	85	—21			12.5	9.0	—3.5	
Basal	Basal	123	108	96	—12	98	125	12.7	9.8	—2.9	12.1
Basal	Extra	119	108	109	1	90	118	11.1	7.9	—3.2	11.8
Basal	Extra	120	116	100	—16			13.5	10.0	—3.5	
Extra	Basal	129	98	87	—11	83	102	15.4	10.8	—4.6	13.1
Extra	Basal	130	90	76	—14			14.2	11.6	—2.6	
Extra	Extra	126	116	102	—14			15.5	11.6	—3.9	
Extra	Extra	127	98	84	—14	86	100	13.0	9.3	—3.7	12.0
		<i>m</i>	105.0	92.4	—12.6			13.49	10.00	—3.49	
CALORIC GROUP L											
Basal	Basal	22	93	90	—3			13.0	9.6	—3.4	
Basal	Basal	23	118	113	—5	124	130	12.8	9.6	—3.2	12.5
Basal	Extra	19	106	88	—18			14.8	9.6	—5.2	
Basal	Extra	20	106	108	2			12.3	8.1	—4.2	
Extra	Basal	29	89	86	—3			15.8	12.0	—3.8	
Extra	Basal	30	112	91	—21			12.5	9.1	—3.4	
Extra	Extra	26	98	104	6	96	100	14.3	9.1	—5.2	14.0
Extra	Extra	27	88	86	—2			16.8	11.7	—5.1	
		<i>m</i>	101.2	95.8	—5.4			14.04	9.85	—4.19	
CALORIC GROUP G											
Basal	Basal	4	95	77	—18	91	86	12.7	10.9	—1.8	13.0
Basal	Basal	5	133	117	—16			12.8	10.4	—2.4	
Basal	Extra	1	104	94	—10			14.7	9.4	—5.3	
Basal	Extra	2	120	80	—40	98	103	12.9	11.9	—1.0	13.9
Extra	Basal	11	108	106	—2			11.9	10.0	—1.9	
Extra	Basal	12	104	89	—15			17.2	12.2	—5.0	
Extra	Extra	8	106	116	10			12.6	8.6	—4.0	
Extra	Extra	9	83	74	—9			17.5	11.8	—5.7	
		<i>m</i>	106.6	94.1	—12.5			14.04	10.65	—3.39	
CALORIC GROUP T											
Basal	Basal	104	93	93	0	82	104	13.4	8.6	—4.8	13.9
Basal	Basal	105	117	103	—14			12.6	9.2	—3.4	
Basal	Extra	101	121	110	—11	103	106	10.3	8.3	—2.0	11.6
Basal	Extra	102	123	82	—41			12.2	11.2	—1.0	
Extra	Basal	111	108	79	—29			12.4	10.1	—2.3	
Extra	Basal	112	116	117	1	120	116	11.8	9.1	—2.7	12.1
Extra	Extra	108	94	102	8			13.7	9.7	—4.0	
Extra	Extra	109	115	101	—14	93	106	14.0	11.2	—2.8	12.9
		<i>m</i>	110.9	98.4	—12.5			12.55	9.68	—2.87	
		<i>M</i>	105.9	95.2	—10.7			13.53	10.04	—3.49	
		<i>SD</i>	11.8	12.8	12.0			1.71	1.24	1.25	

TABLE 495

RESPIRATORY QUOTIENT (columns 1 to 3) and RESPIRATORY EFFICIENCY, in cc. of oxygen removed per liter of ventilation (columns 4 to 9). Both functions were measured during the last 5 minutes of a 30-minute walk on a motor-driven treadmill at 3.5 miles per hour and a 10 per cent grade.

Supplement			1	2	3	4	5	6	7	8	9
Protein	Vitamin	Subject	Period								
			C	S24	dS24	C	S24	dS24	R6	R12	R20
CALORIC GROUP Z											
Basal	Basal	122	0.89	0.93	0.04	59.5	57.0	—2.5	56.1	58.4	
Basal	Basal	123	0.92	0.95	0.03	57.6	41.7	—15.9	46.2		58.8
Basal	Extra	119	0.88	0.92	0.04	55.2	47.1	—8.1	49.3	53.5	49.9
Basal	Extra	120	0.86	0.88	0.02	63.6	58.7	—4.9	56.1		
Extra	Basal	129	0.94	0.94	0.00	47.7	46.5	—1.2	46.7		49.0
Extra	Basal	130	0.86	0.89	0.03	54.4	47.9	—6.5	48.6	50.8	
Extra	Extra	126	0.89	0.90	0.01	53.8	46.4	—7.4	48.8	50.6	
Extra	Extra	127	0.87	0.94	0.07	54.2	42.0	—12.2	49.3		50.1
<i>m</i>			0.889	0.919	0.030	55.75	48.41	—7.34	50.14		
CALORIC GROUP L											
Basal	Basal	22	0.91	0.93	0.02	59.6	54.1	—5.5	57.8		
Basal	Basal	23	0.82	0.91	0.09	54.8	44.6	—10.2	45.7	54.5	52.5
Basal	Extra	19	0.87	0.97	0.10	62.3	50.4	—11.9	52.6	54.2	
Basal	Extra	20	0.91	0.95	0.04	54.4	40.1	—14.3	44.8		
Extra	Basal	29	0.87	0.95	0.08	55.4	39.0	—16.4	49.0		
Extra	Basal	30	0.91	0.97	0.06	51.0	41.9	—9.1	47.2	48.3	
Extra	Extra	26	0.88	1.00	0.12	54.7	44.3	—10.4	46.7	51.7	53.0
Extra	Extra	27	0.85	0.90	0.05	58.8	51.4	—7.66	50.6		
<i>m</i>			0.878	0.948	0.070	56.38	45.72	—10.6	49.30		
CALORIC GROUP G											
Basal	Basal	4	0.83	0.93	0.10	63.5	53.3	—10.2	55.4	56.1	58.0
Basal	Basal	5	0.89	0.90	0.01	48.4	45.4	—3.0	44.7		
Basal	Extra	1	0.86	0.94	0.08	52.6	49.1	—3.5	48.6		
Basal	Extra	2	0.87	0.92	0.05	57.2	47.3	—9.9	49.8	51.1	51.3
Extra	Basal	11	0.89	0.88	—0.01	57.5	50.3	—7.2	51.0		
Extra	Basal	12	0.86	0.98	0.12	53.8	44.1	—9.7	50.0	52.4	
Extra	Extra	8	0.89	1.00	0.11	56.1	35.6	—20.5	48.9		
Extra	Extra	9	0.85	0.96	0.11	55.2	42.6	—12.6	47.4	47.5	
<i>m</i>			0.868	0.939	0.071	55.54	45.96	—9.58	49.48		
CALORIC GROUP T											
Basal	Basal	104	0.85	0.95	0.10	55.1	46.9	—8.2	47.9	49.6	52.4
Basal	Basal	105	0.88	0.97	0.09	50.0	36.3	—13.7	45.1		
Basal	Extra	101	0.90	0.96	0.06	57.3	42.7	—14.6	51.3		58.2
Basal	Extra	102	0.88	0.93	0.05	53.4	46.7	—6.7	47.8	51.9	
Extra	Basal	111	0.88	0.91	0.03	51.6	46.4	—5.2	41.0		
Extra	Basal	112	0.87	0.84	—0.03	55.4	50.2	—5.2	52.4	54.2	49.4
Extra	Extra	108	0.91	0.96	0.05	49.8	44.2	—5.6	47.0		
Extra	Extra	109	0.86	0.89	0.03	63.4	52.6	—10.8	53.4	53.2	54.6
<i>m</i>			0.879	0.926	0.047	54.5	45.75	—8.75	48.24		
<i>M</i>			0.878	0.933	0.055	55.54	46.46	—9.08	49.29		
<i>SD</i>			0.026	0.036	0.040	4.14	5.44	4.46			

TABLE 496

OXYGEN CONSUMPTION, in cc. per minute during the last 5 minutes of a 30-minute walk on a motor-driven treadmill at 3.5 miles per hour and a 10 per cent grade.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R12	R20
CALORIC GROUP Z							
Basal	Basal	122	1790	1140	—650	1296	
Basal	Basal	123	1635	1332	—303		1827
Basal	Extra	119	1668	1210	—458	1237	1884
Basal	Extra	120	1861	1295	—566		
Extra	Basal	129	1817	1282	—535		1875
Extra	Basal	130	1737	1320	—417	1367	
Extra	Extra	126	2164	1548	—616	1614	
Extra	Extra	127	1623	1156	—467		1721
		<i>m</i>	1786.9	1285.4	—501.5		
CALORIC GROUP L							
Basal	Basal	22	1672	1244	—428		
Basal	Basal	23	1840	1401	—439	1471	1940
Basal	Extra	19	1825	1244	—581	1353	
Basal	Extra	20	1662	1271	—391		
Extra	Basal	29	1838	1480	—358		
Extra	Basal	30	1610	1139	—471	1252	
Extra	Extra	26	1842	1301	—541	1447	2058
Extra	Extra	27	1968	1455	—513		
		<i>m</i>	1782.1	1316.9	—465.2		
CALORIC GROUP G							
Basal	Basal	4	1532	1176	—356	1403	1784
Basal	Basal	5	2127	1554	—573		
Basal	Extra	1	1965	1207	—758		
Basal	Extra	2	1855	1453	—402	1525	2044
Extra	Basal	11	1620	1306	—314	1774	
Extra	Basal	12	2230	1584	—646		
Extra	Extra	8	1614	1270	—344		
Extra	Extra	9	1906	1348	—558	1514	
		<i>m</i>	1856.1	1362.2	—493.9		
CALORIC GROUP T							
Basal	Basal	104	1699	1197	—502	1418	1945
Basal	Basal	105	1883	1391	—492		
Basal	Extra	101	1634	1253	—381		1840
Basal	Extra	102	1750	1328	—422	1673	
Extra	Basal	111	1625	1244	—381		
Extra	Basal	112	1734	1416	—318	1564	1946
Extra	Extra	108	1750	1428	—322		
Extra	Extra	109	2044	1546	—498	1695	2018
		<i>m</i>	1764.9	1350.4	—414.5		
		<i>M</i>	1797.5	1328.7	—468.8		
		<i>SD</i>	173.9	127.3			

TABLE 497

ENERGY COST OF AEROBIC WORK, as gm. calories used per kg. of body weight per minute during the last 5 minutes of a 30-minute walk on a motor-driven treadmill at 3.5 miles per hour and a 10 per cent grade.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	R6	R12
CALORIC GROUP Z							
Basal	Basal	122	115.5	106.1	—9.4	110.7	112.2
Basal	Basal	123	108.9	116.4	7.5	113.7	
Basal	Extra	119	106.9	103.2	—3.7	104.9	101.4
Basal	Extra	120	112.2	109.6	—2.6	114.0	
Extra	Basal	129	119.1	106.4	—12.7	116.0	
Extra	Basal	130	112.1	108.7	—3.4	114.7	109.7
Extra	Extra	126	112.1	110.9	—1.2	110.9	116.2
Extra	Extra	127	104.8	103.8	—1.0	107.3	
		<i>m</i>	111.45	108.1	—3.31	111.52	
CALORIC GROUP L							
Basal	Basal	22	111.5	112.8	1.3	108.1	
Basal	Basal	23	110.7	119.6	8.9	116.1	113.5
Basal	Extra	19	110.1	107.2	—2.9	109.7	110.7
Basal	Extra	20	111.0	117.3	6.3	114.7	
Extra	Basal	29	108.9	121.8	12.9	111.8	
Extra	Basal	30	101.2	96.4	—4.8	102.8	
Extra	Extra	26	109.4	104.6	—4.8	105.6	109.0
Extra	Extra	27	111.2	116.8	5.6	113.0	
		<i>m</i>	109.25	112.06	2.81	110.22	
CALORIC GROUP G							
Basal	Basal	4	102.8	108.8	6.0	101.4	106.8
Basal	Basal	5	113.9	118.7	4.8	115.9	
Basal	Extra	1	108.2	93.8	—14.4	106.9	
Basal	Extra	2	107.8	111.8	4.4	112.7	110.8
Extra	Basal	11	106.4	114.4	8.0	113.6	
Extra	Basal	12	117.6	117.2	—0.4	115.5	119.3
Extra	Extra	8	105.7	119.1	13.4	111.0	
Extra	Extra	9	110.5	107.8	—2.7	105.7	106.2
		<i>m</i>	109.11	111.45	2.34	110.34	
CALORIC GROUP T							
Basal	Basal	104	105.4	102.5	—2.9	103.8	103.8
Basal	Basal	105	115.1	125.9	10.8	118.8	
Basal	Extra	101	108.5	115.5	7.0	117.4	
Basal	Extra	102	109.2	112.7	3.5	110.0	117.5
Extra	Basal	111	109.0	112.3	3.3	117.2	
Extra	Basal	112	121.2	124.0	2.8	117.7	116.7
Extra	Extra	108	110.7	113.9	3.2	115.8	
Extra	Extra	109	110.5	115.2	4.7	110.3	115.7
		<i>m</i>	111.20	115.25	4.05	113.88	
		<i>M</i>	110.25	111.72	1.47	111.49	
		<i>SD</i>	4.36	7.44			

TABLE 498

MAXIMUM WORK TEST, TIME OF RUN in seconds. The work on a motor-driven treadmill (speed = 7 miles per hour, grade = 8.6 per cent) was carried to exhaustion or to a maximum of 5 minutes.

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	300	139	69	—231	—7	71	152
Basal	Basal	123	300	106	17	—283	4	35	
Basal	Extra	119	249	152	90	—159	—4	5	109
Basal	Extra	120	172	124	82	—90	3	19	99
Extra	Basal	129	152	42	29	—123	—2	33	
Extra	Basal	130	300	125	42	—258	—1	21	300
Extra	Extra	126	184	97	33	—151	6	29	
Extra	Extra	127	300	121	59	—241	9	63	
		<i>m</i>	244.6	113.2	52.6	—192.0	2.75	34.50	
CALORIC GROUP L									
Basal	Basal	22	300	147	65	—235	17	69	247
Basal	Basal	23	262	127	105	—157	19	93	
Basal	Extra	19	300	87	19	—281	20	115	300
Basal	Extra	20	300	116	18	—282	4	26	
Extra	Basal	29	234	23	12	—222	12	69	300
Extra	Basal	30	255	96	45	—210	22	64	
Extra	Extra	26	300	145	99	—201	41	109	
Extra	Extra	27	300	129	52	—248	20	54	
		<i>m</i>	281.4	108.8	51.9	—229.5	19.38	74.88	
CALORIC GROUP G									
Basal	Basal	4	300	148	120	—180	49	78	232
Basal	Basal	5	104	92	60	—44	12	29	129
Basal	Extra	1	214	81	29	—185	23	53	
Basal	Extra	2	172	100	40	—132	15	34	
Extra	Basal	11	180	138	79	—101	34	77	300
Extra	Basal	12	176	113	10	—166	27	92	
Extra	Extra	8	288	40	19	—269	30	97	145
Extra	Extra	9	300	87	43	—257	48	119	
		<i>m</i>	216.8	99.9	50.0	—166.8	29.8	72.4	
CALORIC GROUP T									
Basal	Basal	104	300	107	37	—263	70	133	289
Basal	Basal	105	207	103	27	—180	52	102	200
Basal	Extra	101	198	130	25	—173	48	99	
Basal	Extra	102	134	94	57	—77	25	64	300
Extra	Basal	111	252	78	32	—220	29	58	
Extra	Basal	112	300	127	102	—198	24	56	
Extra	Extra	108	263	85	31	—232	45	71	145
Extra	Extra	109	134	98	60	—74	37	65	
		<i>m</i>	223.5	102.8	46.4	—177.1	41.2	81.0	
		<i>M</i>	241.6	106.2	50.2	—193.1	27.7	65.7	
		<i>SD</i>	62.9		30.0				

TABLE 499
MAXIMUM WORK TEST, "Harvard Fitness Test" score.

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	70	36	23	—47	2	14	40
Basal	Basal	123	78	31	8	—70	0	9	
Basal	Extra	119	62	41	29	—33	0	0	
Basal	Extra	120	40	29	23	—17	3	6	
Extra	Basal	129	45	18	12	—33	0	9	30
Extra	Basal	130	80	43	21	—59	—2	2	
Extra	Extra	126	49	32	14	—35	5	11	72
Extra	Extra	127	78	36	19	—59	4	17	
		<i>m</i>	62.8	33.2	18.6	—44.1	1.5	8.5	
CALORIC GROUP L									
Basal	Basal	22	82	43	22	—60	6	20	57
Basal	Basal	23	61	34	27	—34	5	22	
Basal	Extra	19	82	30	9	—73	7	29	
Basal	Extra	20	72	32	6	—66	1	7	
Extra	Basal	29	63	10	8	—55	2	18	76
Extra	Basal	30	65	32	17	—48	7	17	
Extra	Extra	26	82	49	35	—47	21	28	
Extra	Extra	27	88	41	21	—67	10	16	
		<i>m</i>	74.4	33.9	18.1	—56.3	7.4	19.6	
CALORIC GROUP G									
Basal	Basal	4	83	47	39	—44	16	17	67
Basal	Basal	5	26	22	19	—7	3	7	
Basal	Extra	1	66	41	17	—49	16	26	34
Basal	Extra	2	44	32	18	—26	0	6	
Extra	Basal	11	48	40	26	—22	10	19	
Extra	Basal	12	52	34	5	—47	11	26	
Extra	Extra	8	70	14	7	—63	13	26	
Extra	Extra	9	90	33	18	—72	19	33	
		<i>m</i>	59.9	32.9	18.6	—41.2	11.0	20.0	
CALORIC GROUP T									
Basal	Basal	104	84	35	18	—66	24	34	65
Basal	Basal	105	51	33	10	—41	15	26	52
Basal	Extra	101	48	33	8	—40	18	27	
Basal	Extra	102	36	27	22	—14	6	12	
Extra	Basal	111	62	27	13	—49	10	17	66
Extra	Basal	112	77	39	28	—49	14	11	
Extra	Extra	108	76	30	18	—58	15	14	
Extra	Extra	109	42	34	20	—22	17	22	
		<i>m</i>	59.5	32.2	17.1	—42.3	14.9	20.3	42
		<i>M</i>	64.1	33.1	18.1	—46.0	8.7	17.1	
		<i>SD</i>	17.2	8.6	8.3	17.9	7.2	9.0	

TABLE 500

VISUAL ACUITY, in mm., as the diameter of a solid black circle of threshold size at a distance of 15 ft.; the illumination intensity at the level of the target was 1 foot-candle (column 1) and 100 foot-candles (column 2). FUSION FREQUENCY, in flickers per second (column 3). HEARING LOSS, in decibels, measured by the Maico D-5 audiometer at the frequencies of 128, 512, 4096, and 8192 cycles per second (columns 4, 5, 6, and 7). All values are for the control period.

Supplement		Subject	1	2	3	4	5	6	7
Protein	Vitamin								
CALORIC GROUP Z									
Basal	Basal	122	2.0	0.67	36.5	7.7	-1.7	15.3	-4.0
Basal	Basal	123	2.0	0.69	35.0	8.3	0.7	54.0	13.0
Basal	Extra	119	2.0	0.75	36.5	4.0	-1.0	6.7	-6.7
Basal	Extra	120	2.0	0.62	32.0	7.3	7.3	-4.0	2.7
Extra	Basal	129	3.5	0.88	36.0	2.0	-2.7	3.7	3.3
Extra	Basal	130	1.6	0.50	38.0	3.3	3.3	2.3	-5.7
Extra	Extra	126	2.8	1.00	38.0	7.7	3.0	12.7	-2.7
Extra	Extra	127	1.9	0.50	33.0	4.3	-4.3	13.3	38.0
		<i>m</i>	2.22	0.701	35.62	5.58	0.58	13.00	4.74
CALORIC GROUP L									
Basal	Basal	22	1.6	0.50	36.5	4.3	-9.0	-8.3	-7.0
Basal	Basal	23	1.9	0.69	33.5	3.3	13.3	16.3	-3.0
Basal	Extra	19	2.1	0.62	40.5	13.3	7.3	14.7	2.7
Basal	Extra	20	2.0	0.71	41.0	4.0	3.7	-7.7	-5.0
Extra	Basal	29	2.0	0.73	39.0	-4.3	-3.3	-10.0	-9.3
Extra	Basal	30	2.1	0.94	31.5	-3.0	-3.0	-2.7	-4.0
Extra	Extra	26	2.5	0.75	34.0	2.0	-2.7	-8.7	-3.0
Extra	Extra	27	2.1	0.81	39.5	-1.3	-4.7	-7.0	-6.7
		<i>m</i>	2.04	0.719	36.94	2.29	0.20	-1.68	-4.41
CALORIC GROUP G									
Basal	Basal	4	2.2	0.67	29.5	3.0	-7.0	-6.0	12.7
Basal	Basal	5	2.5	0.75	37.5	11.7	2.7	-10.0	-2.7
Basal	Extra	1	2.4	0.94	39.5	3.0	-2.7	-10.0	-10.0
Basal	Extra	2	3.0	0.88	34.0	7.3	3.3	16.0	-4.3
Extra	Basal	11	2.1	0.67	37.0	3.0	-7.7	3.0	-4.0
Extra	Basal	12	1.8	0.54	33.5	0.7	-4.3	46.7	-6.7
Extra	Extra	8	1.8	0.75	33.5	3.0	3.0	-7.7	-5.3
Extra	Extra	9	1.8	0.50	35.5	3.7	-2.7	-7.0	7.3
		<i>m</i>	2.20	0.712	35.00	4.42	-1.92	3.12	-1.62
CALORIC GROUP T									
Basal	Basal	104	1.9	0.58	39.0	3.3	3.0	-7.7	-7.0
Basal	Basal	105	1.6	0.67	38.0	4.3	-3.0	-9.3	-7.7
Basal	Extra	101	2.8	0.71	40.0	4.7	2.0	27.3	18.0
Basal	Extra	102	1.8	0.67	31.0	2.7	-3.7	-8.0	-7.7
Extra	Basal	111	1.4	0.50	33.5	3.7	4.0	3.0	-4.0
Extra	Basal	112	1.8	0.50	40.0	3.0	-2.0	-4.7	-10.0
Extra	Extra	108	2.5	0.54	40.5	2.3	-1.3	3.0	-6.0
Extra	Extra	109	2.0	0.69	39.5	5.6	-5.0	0.7	-7.3
		<i>m</i>	1.98	0.607	37.69	3.71	0.50	0.13	-3.96
		<i>M</i>	2.11	0.685	36.31	4.00	-0.16	3.75	-1.31
		<i>SD</i>	0.45	0.140	3.17	4.63	4.84	15.72	9.92

TABLE 501

HEARING LOSS, in decibels, measured by the Maico D-5 audiometer at the frequency of 2048 cycles per second.

Supplement		Subject	Period				
Protein	Vitamin		C	S24	dS24	△R6	△R12
CALORIC GROUP Z							
Basal	Basal	122	3.0	—0.7	—3.7	—4.3	8.0
Basal	Basal	123	18.3	12.7	—5.6	3.6	10.0
Basal	Extra	119	4.3	—7.0	—11.3	—3.0	7.0
Basal	Extra	120	—4.7	—7.0	—2.3	—3.0	—3.0
Extra	Basal	129	0.0	—3.0	—3.0	6.0	0.7
Extra	Basal	130	—3.0	—7.7	—4.7	11.0	6.4
Extra	Extra	126	—3.0	—7.3	—4.3	—2.7	0.3
Extra	Extra	127	—3.0	—10.0	—7.0	0.0	4.0
		<i>m</i>	1.49	—3.75	—5.24	0.95	4.18
CALORIC GROUP L							
Basal	Basal	22	0.3	—6.0	—6.3	—1.7	4.7
Basal	Basal	23	8.0	7.3	—0.7	4.7	9.7
Basal	Extra	19	3.0	—3.0	—6.0	—2.7	6.0
Basal	Extra	20	—8.0	—3.3	4.7	0.3	6.3
Extra	Basal	29	—6.7	—10.0	—3.3	17.0	3.7
Extra	Basal	30	4.3	3.0	—1.3	9.7	14.3
Extra	Extra	26	—3.0	—7.7	—4.7	0.0	4.7
Extra	Extra	27	—4.0	—10.0	—6.0	0.0	4.3
		<i>m</i>	—0.76	—3.71	—2.95	3.41	6.71
CALORIC GROUP G							
Basal	Basal	4	1.3	—7.7	—9.0	15.0	10.0
Basal	Basal	5	3.0	—5.0	—8.0	—2.0	8.7
Basal	Extra	1	1.7	—6.7	—8.4	13.7	10.0
Basal	Extra	2	5.7	—2.0	—7.7	5.0	9.0
Extra	Basal	11	3.0	—3.3	—6.3	0.3	—4.4
Extra	Basal	12	—6.0	—2.7	3.3	—7.3	—0.3
Extra	Extra	8	—2.3	—2.7	—0.4	5.7	5.7
Extra	Extra	9	—3.0	—7.7	—4.7	0.7	2.7
		<i>m</i>	0.43	—4.72	—5.15	3.89	5.18
CALORIC GROUP T							
Basal	Basal	104	3.3	—7.7	—11.0	0.4	4.7
Basal	Basal	105	—5.0	—7.7	—2.7	—2.3	9.0
Basal	Extra	101	12.7	—1.3	—14.0	4.3	13.6
Basal	Extra	102	—3.7	—7.3	—3.6	1.0	4.3
Extra	Basal	111	—4.0	—6.7	—2.7	—3.3	0.0
Extra	Basal	112	—5.7	—7.3	—1.6	0.6	4.3
Extra	Extra	108	8.7	—2.7	—11.4	10.0	10.0
Extra	Extra	109	3.0	—1.3	—4.3	4.3	9.6
		<i>m</i>	1.16	—5.25	—6.41	1.88	6.94
		<i>M</i>	0.58	—4.36	—4.94	2.53	5.75
		<i>SD</i>	5.89	4.92			

TABLE 502
HAND DYNAMOMETER, in kg.

Supplement			Period							
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20	R32
CALORIC GROUP Z										
Basal	Basal	122	74	55	48	—26	0	4		79
Basal	Basal	123	58	50	39	—19	—1	2	49	55
Basal	Extra	119	51	41	34	—17	6	8	45	48
Basal	Extra	120	53	41	42	—11	—4	—3		
Extra	Basal	129	56	46	49	—7	—3	—1	49	53
Extra	Basal	130	55	48	41	—14	0	2		53
Extra	Extra	126	67	54	45	—22	1	4		
Extra	Extra	127	58	46	40	—18	—2	5	52	60
		<i>m</i>	59.0	47.6	42.2	—16.8	—0.4	2.6		
CALORIC GROUP L										
Basal	Basal	22	65	56	52	—13	—2	0		70
Basal	Basal	23	48	36	38	—10	0	4	46	
Basal	Extra	19	68	50	38	—30	—3	10		62
Basal	Extra	20	60	48	38	—22	0	10		
Extra	Basal	29	59	34	31	—28	4	11		59
Extra	Basal	30	61	50	44	—17	—7	—1		59
Extra	Extra	26	64	51	45	—19	—1	5	58	66
Extra	Extra	27	56	52	51	—5	—3	—4		56
		<i>m</i>	60.1	47.1	42.1	—18.0	—1.5	4.4		
CALORIC GROUP G										
Basal	Basal	4	58	47	43	—15	5	5	53	57
Basal	Basal	5	64	48	43	—21	—1	5		53
Basal	Extra	1	66	54	42	—24	2	15		69
Basal	Extra	2	45	40	36	—9	2	4	40	44
Extra	Basal	11	46	39	38	—8	0	2		45
Extra	Basal	12	59	48	40	—19	2	7		
Extra	Extra	8	48	34	27	—21	3	15		
Extra	Extra	9	55	44	40	—15	4	9		62
		<i>m</i>	55.1	44.2	38.6	—16.5	2.1	7.7		
CALORIC GROUP T										
Basal	Basal	104	68	56	50	—18	4	8	65	
Basal	Basal	105	70	60	50	—20	4	8		
Basal	Extra	101	50	45	33	—17	3	14	52	
Basal	Extra	102	45	41	38	—7	—2	3		
Extra	Basal	111	55	49	46	—9	3	11		
Extra	Basal	112	64	53	52	—12	—3	—2	61	62
Extra	Extra	108	55	44	36	—19	7	10		
Extra	Extra	109	60	49	48	—12	—2	2	48	60
		<i>m</i>	58.4	49.6	44.1	—14.3	1.7	6.7		
		<i>M</i>	58.2	47.2	41.8	—16.4	0.5	5.4		
		<i>SD</i>	7.6	6.5	6.3	6.3	3.2	5.1		

TABLE 503
BACK DYNAMOMETER, in kg.

Supplement		Subject	Period						
Protein	Vitamin		C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	174	143	130	—44	—4	0	136
Basal	Basal	123	151	122	109	—42	0	4	
Basal	Extra	119	132	111	107	—25	—8	—4	
Basal	Extra	120	174	151	132	—42	2	0	168
Extra	Basal	130	130	124	98	—32	3	11	
Extra	Extra	126	180	164	157	—23	—12	7	
Extra	Extra	127	189	166	136	—53	—13	—4	168
		<i>m</i>	161.4	140.1	124.1	—37.3	—4.6	2.0	
CALORIC GROUP L									
Basal	Basal	22	201	143	119	—82	—4	13	132
Basal	Basal	23	140	126	108	—32	1	5	
Basal	Extra	19	208	146	119	—89	—17	13	
Basal	Extra	20	168	130	104	—64			214
Extra	Basal	29	170	101	100	—70	15	26	
Extra	Basal	30	149	124	111	—38	—2	—2	
Extra	Extra	26	218	157	147	—71	—7	12	214
Extra	Extra	27	178	130	104	—74	—2	5	
		<i>m</i>	179.0	132.1	114.0	—65.0	—2.3	10.3	
CALORIC GROUP G									
Basal	Basal	4	176	138	108	—68	28	39	168
Basal	Extra	1	178	147	140	—38	0	15	111
Basal	Extra	2	117	113	96	—21	5	11	
Extra	Basal	11	124	94	94	—30	11	11	
Extra	Extra	8	159	105	90	—69	12	29	136
Extra	Extra	9	193	132	132	—61	13	25	
		<i>m</i>	157.8	121.5	110.0	—47.8	11.5	21.7	
CALORIC GROUP T									
Basal	Basal	104	174	145	119	—55	15	28	170
Basal	Basal	105	193	151	119	—74	4	17	145
Basal	Extra	101	138	126	103	—35	6	18	
Basal	Extra	102	122	101	90	—32	2	0	
Extra	Basal	111	178	134	119	—59	2	15	164
Extra	Basal	112	180	147	143	—37	2	2	
Extra	Extra	108	182	147	119	—63	19	30	
Extra	Extra	109	122	101	103	—19	8	17	136
		<i>m</i>	161.1	131.5	114.4	—46.7	7.2	15.9	
		<i>M</i>	149.9	119.3	104.9	—45.1	2.8	12.2	
		<i>SD</i>	55.6		38.2				

TABLE 504

SPEED OF KICK, in thousandths of a second, without load (columns 1 to 4) and with a 10-lb. weight attached to the leg (columns 5 to 8).

			1	2	3	4	5	6	7	8
Supplement			Period							
Protein	Vitamin	Subject	S24	△R6	△R12	R20	S24	△R6	△R12	R20
CALORIC GROUP Z										
Basal	Basal	122	148	2	2		173	—1	1	
Basal	Basal	123	135	4	0	119	179	—10	—18	143
Basal	Extra	119	124	2	—5	105	143	3	4	127
Basal	Extra	120	126	6	5		156	0	—5	
Extra	Basal	129	140	4	3	123	161	14	—9	143
Extra	Basal	130	137	4	—3		225	—51	—71	
Extra	Extra	126	135	—9	—15		167	—22	—27	
Extra	Extra	127	145	5	—15	113	203	—26	—51	127
		<i>m</i>	136.2	2.2	—3.5		175.9	—11.6	—22.0	
CALORIC GROUP L										
Basal	Basal	22	139	—4	5		167	—6	5	
Basal	Basal	23	110	9	7	105	133	11	9	122
Basal	Extra	19	130	—5	—6		199	—47	—42	
Basal	Extra	20	142	—2	—17		205	—21	—52	
Extra	Basal	29	126	1	8		156	3	—1	
Extra	Basal	30	119	5	—1		158	—12	—16	
Extra	Extra	26	135	—6	—4	110	157	0	—2	125
Extra	Extra	27	125	—2	0		152	—3	4	
		<i>m</i>	128.2	—0.5	—1.0		165.9	—9.4	—11.9	
CALORIC GROUP C										
Basal	Basal	4	135	—15	—32	89	181	—52	—65	97
Basal	Basal	5	131	1	—10		163	—4	—12	
Basal	Extra	1	123	1	—8		143	0	—8	
Basal	Extra	2	135	0	—16	119	161	4	—12	139
Extra	Basal	11	121	2	4		151	—7	1	
Extra	Basal	12	165	—6	—28		223	—44	—56	
Extra	Extra	8	159	—7	—26		227	—54	—77	
Extra	Extra	9	133	3	0		165	—13	—8	
		<i>m</i>	137.7	—2.6	—14.5		176.7	—21.2	—29.6	
CALORIC GROUP T										
Basal	Basal	104	146	—5	—19	118	185	—20	—34	127
Basal	Basal	105	122	—8	—14		151	—16	—13	
Basal	Extra	101	144	—11	—20	123	191	—41	—53	150
Basal	Extra	102	126	—7	—9		145	—6	—5	
Extra	Basal	111	127	—2	10		152	11	8	
Extra	Basal	112	132	—4	—11	122	152	—4	—7	141
Extra	Extra	108	149	—36	—35		203	—57	—58	
Extra	Extra	109	112	—6	—11	100	130	—11	—9	116
		<i>m</i>	132.2	—9.9	—13.6		163.6	—18.0	—21.4	
		<i>M</i>	133.6	—2.7	—8.2		170.5	—15.1	—21.2	
		<i>SD</i>	12.3	8.2	11.9		26.6	20.8	25.8	

TABLE 505

TAPPING, as the number of taps per 10 seconds (column 1). BALL-PIPE TEST SCORE, as the number of balls passed through a vertical pipe in 1 minute (column 2). GROSS BODY REACTION TIME, in hundredths of a second (column 3). PATTERN-TRACING, number of errors (column 4).
All values are for the control period.

Supplement		Subject	1	2	3	4
Protein	Vitamin					
CALORIC GROUP Z						
Basal	Basal	122	66	76	37	104
Basal	Basal	123	72	78	42	127
Basal	Extra	119	71	68	40	104
Basal	Extra	120	61	70	46	152
Extra	Basal	129	69	81	42	122
Extra	Basal	130	51	69	48	138
Extra	Extra	126	72	74	41	115
Extra	Extra	127	64	82	38	108
		<i>m</i>	65.7	74.8	41.7	121.2
CALORIC GROUP L						
Basal	Basal	22	68	85	38	102
Basal	Basal	23	62	72	45	105
Basal	Extra	19	58	78	38	122
Basal	Extra	20	72	80	43	98
Extra	Basal	29	64	79	38	140
Extra	Basal	30	60	70	40	121
Extra	Extra	26	73	79	44	118
Extra	Extra	27	74	84	38	96
		<i>m</i>	66.4	78.4	40.5	112.7
CALORIC GROUP G						
Basal	Basal	4	60	83	43	127
Basal	Basal	5	58	77	46	123
Basal	Extra	1	73	68	41	109
Basal	Extra	2	69	68	42	123
Extra	Basal	11	62	72	45	111
Extra	Basal	12	65	72	47	123
Extra	Extra	8	76	80	39	117
Extra	Extra	9	67	66	41	105
		<i>m</i>	66.2	73.2	43.0	117.2
CALORIC GROUP T						
Basal	Basal	104	66	72	40	107
Basal	Basal	105	68	70	47	101
Basal	Extra	101	65	83	45	110
Basal	Extra	102	67	80	39	124
Extra	Basal	111	68	77	42	108
Extra	Basal	112	69	66	45	132
Extra	Extra	108	71	79	42	108
Extra	Extra	109	69	73	53	91
		<i>m</i>	67.9	75.0	44.1	110.1
		<i>M</i>	66.6	75.3	42.3	115.3
		<i>SD</i>	5.5	5.7	3.7	13.7

TABLE 506
PATTERN-TRACING, total length of the contact errors, in tenths of a second.

Supplement			Period						
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20
CALORIC GROUP Z									
Basal	Basal	122	28	28	32	4	—2	—3	
Basal	Basal	123	38	38	39	1	4	—4	40
Basal	Extra	119	32	37	35	3	—4	—7	30
Basal	Extra	120	44	51	49	5	—4	—2	
Extra	Basal	129	44	52	47	3	—3	—3	41
Extra	Basal	130	49	54	61	12	—7	—2	
Extra	Extra	126	35	40	47	12	—7	—9	
Extra	Extra	127	34	37	40	6	—3	—2	32
		<i>m</i>	38.0	42.1	43.7	5.7	—3.2	—4.0	
CALORIC GROUP L									
Basal	Basal	22	27	35	32	5	—2	—6	
Basal	Basal	23	34	39	37	3	—4	0	35
Basal	Extra	19	46	55	52	6	—2	0	
Basal	Extra	20	26	33	35	9	—1	—2	
Extra	Basal	29	34	38	37	3	1	—6	
Extra	Basal	30	40	41	50	10	—8	—10	
Extra	Extra	26	38	42	46	8	—7	—2	40
Extra	Extra	27	28	26	29	1	—3	—2	
		<i>m</i>	34.1	38.6	39.7	5.6	—3.2	—3.5	
CALORIC GROUP G									
Basal	Basal	4	42	41	50	8	—8	—6	44
Basal	Basal	5	44	40	44	0	—3	0	
Basal	Extra	1	33	41	40	7	—4	—5	
Basal	Extra	2	55	58	58	3	—2	—1	58
Extra	Basal	11	35	39	36	1	—1	0	
Extra	Basal	12	46	50	51	5	1	7	
Extra	Extra	8	33	44	40	7	—1	—2	
Extra	Extra	9	28	39	38	10	—6	—9	
		<i>m</i>	39.5	44.0	44.6	5.1	—3.0	—2.0	
CALORIC GROUP T									
Basal	Basal	104	33	35	35	2	—1	—6	34
Basal	Basal	105	28	40	35	7	—1	—1	
Basal	Extra	101	35	43	43	8	—6	—6	44
Basal	Extra	102	32	35	31	—1	—4	—1	
Extra	Basal	111	30	40	40	10	—9	—9	
Extra	Basal	112	52	57	67	15	—10	—8	53
Extra	Extra	108	42	52	57	15	—10	—11	
Extra	Extra	109	31	34	37	6	—6	—3	32
		<i>m</i>	35.4	42.0	43.1	7.7	—5.9	—5.6	
		<i>M</i>	36.7	41.7	42.8	6.1	—3.8	—3.8	
		<i>SD</i>	7.7	8.1	9.4	3.1	3.3	3.8	

TABLE 507

CAVD TEST OF INTELLIGENCE, levels M, N, O, P, and Q. Score for: Completions (column 1), Arithmetical Problems (column 2), Vocabulary (column 3), Directions (column 4), and "Altitude" (column 5). Army standard scores in the ARMY GENERAL CLASSIFICATION TEST (column 6). All values are for the control period.

Supplement		Subject	1	2	3	4	5	6
Protein	Vitamin							
CALORIC GROUP Z								
Basal	Basal	122	37	46	32	37	430	146
Basal	Basal	123	45	50	40	41	444	154
Basal	Extra	119	28	38	40	43	431	138
Basal	Extra	120	31	43	17	30	424	127
Extra	Basal	129	42	45	40	34	435	147
Extra	Basal	130	23	16	29	26	412	125
Extra	Extra	126	36	41	28	39	427	144
Extra	Extra	127	30	43	30	38	426	142
		<i>m</i>	34.0	40.2	32.0	36.0	428.6	140.4
CALORIC GROUP L								
Basal	Basal	22	36	50	32	39	435	151
Basal	Basal	23	40	48	30	44	435	142
Basal	Extra	19	29	27	30	36	417	122
Basal	Extra	20	39	40	40	39	434	141
Extra	Basal	29	33	36	31	28	422	128
Extra	Basal	30	21	4	26	35	404	123
Extra	Extra	26	22	35	13	27	411	128
Extra	Extra	27	22	37	25	32	418	135
		<i>m</i>	30.2	34.6	28.4	35.0	422.0	133.8
CALORIC GROUP G								
Basal	Basal	4	35	47	37	36	432	141
Basal	Basal	5	37	35	29	39	418	131
Basal	Extra	1	38	46	31	38	434	146
Basal	Extra	2	39	42	29	35	429	135
Extra	Basal	11	35	50	33	36	430	147
Extra	Basal	12	35	46	39	38	433	150
Extra	Extra	8	28	47	15	32	424	136
Extra	Extra	9	31	42	31	31	426	146
		<i>m</i>	34.8	44.4	30.5	35.6	428.2	141.5
CALORIC GROUP T								
Basal	Basal	104	34	41	25	35	426	141
Basal	Basal	105	13	33	6	25	402	135
Basal	Extra	101	36	48	34	40	432	141
Basal	Extra	102	32	42	29	39	430	133
Extra	Basal	111	47	48	44	42	454	147
Extra	Basal	112	30	45	27	39	424	141
Extra	Extra	108	19	20	25	29	407	132
Extra	Extra	109	40	46	35	38	433	144
		<i>m</i>	31.4	40.4	28.1	35.9	426.0	139.2
		<i>M</i>	32.6	39.9	29.8	35.6	426.2	138.7
		<i>SD</i>	7.7	10.4	8.3	5.0	11.1	8.5

TABLE 508

BATTERY OF INTELLECTIVE TESTS: Flags (column 1), First Letters (column 2), Number Identities (column 3), Word-Number Recall (column 4), Multiplications (column 5), and Letter Series (column 6). All values are for the control period.

Supplement		Subject	1	2	3	4	5	6
Protein	Vitamin							
CALORIC GROUP Z								
Basal	Basal	122	81	38	68	40	40	22
Basal	Basal	123	66	52	64	28	76	22
Basal	Extra	119	34	41	35	23	44	15
Basal	Extra	120	43	32	38	25	42	12
Extra	Basal	129	60	43	32	50	16	18
Extra	Basal	130	58	46	22	28	19	14
Extra	Extra	126	70	39	30	34	28	16
Extra	Extra	127	82	65	68	39	57	24
		<i>m</i>	61.8	44.5	44.6	33.4	40.2	17.9
CALORIC GROUP L								
Basal	Basal	22	47	49	47	26	52	16
Basal	Basal	23	50	34	26	32	50	12
Basal	Extra	19	68	40	58	42	32	10
Basal	Extra	20	62	54	44	14	44	16
Extra	Basal	29	62	52	50	16	72	10
Extra	Basal	30	34	42	49	18	41	7
Extra	Extra	26	64	46	46	20	49	19
Extra	Extra	27	62	52	50	18	54	9
		<i>m</i>	56.1	46.1	46.2	23.2	49.2	12.4
CALORIC GROUP G								
Basal	Basal	4	56	51	43	33	44	20
Basal	Basal	5	34	42	29	35	27	13
Basal	Extra	1	58	36	38	22	32	18
Basal	Extra	2	56	46	52	36	51	16
Extra	Basal	11	52	49	40	11	48	15
Extra	Basal	12	54	50	40	11	22	16
Extra	Extra	8	76	40	46	26	31	14
Extra	Extra	9	80	40	64	30	56	22
		<i>m</i>	58.2	44.2	44.0	25.5	38.9	16.8
CALORIC GROUP T								
Basal	Basal	104	60	40	41	41	50	16
Basal	Basal	105	58	36	38	14	30	10
Basal	Extra	101	68	55	38	24	58	23
Basal	Extra	102	41	46	36	12	34	8
Extra	Basal	111	60	65	39	43	58	20
Extra	Basal	112	58	60	38	32	28	15
Extra	Extra	108	54	42	35	18	60	14
Extra	Extra	109	42	46	44	26	44	10
		<i>m</i>	55.1	48.8	38.6	26.2	45.2	14.5
		<i>M</i>	57.8	45.9	43.4	27.1	43.4	15.4
		<i>SD</i>	12.9	8.2	11.6	10.4	14.6	4.6

TABLE 509

COMPOSITE RATINGS OF THE COMPLAINTS as the sum of the scores for twenty-one items. Each item was evaluated by the subject on a scale from +5 (extreme deterioration) through 0 (normal) to -5 (much better than normal).

Supplement			Period							
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20	R32
CALORIC GROUP Z										
Basal	Basal	122	0	30	44	44	—14	—29	4	0
Basal	Basal	123	0	25	20	20	—11	—19	0	1
Basal	Extra	119	0	15	39	39	—14	—21	2	0
Basal	Extra	120	0	36	41	41	—14	—20	5	
Extra	Basal	129	0	21	31	31	—31	—38	15	
Extra	Basal	130	0	43	45	45	—21	—27	2	—2
Extra	Extra	126	0	31	51	51	—25	—27	—3	
Extra	Extra	127	0	11	15	15	—2	—12	0	0
		<i>m</i>	0	26.5	35.8	35.8	—16.5	—24.1	3.1	
CALORIC GROUP L										
Basal	Basal	22	0	47	58	58	—13	—33	4	3
Basal	Basal	23	0	9	16	16	—2	—8	4	
Basal	Extra	19	0	31	23	23	—24	—23	1	3
Basal	Extra	20	0	21	43	43	9	—11	16	1
Extra	Basal	29	0	38	32	32	—6	—11	—9	—3
Extra	Basal	30	0	23	50	50	—20	—31	0	0
Extra	Extra	26	0	21	37	37	—22	—33	0	0
Extra	Extra	27	1	1	11	10	—7	—7	1	—1
		<i>m</i>	0.1	23.9	33.7	33.6	—10.6	—19.6	2.1	
CALORIC GROUP G										
Basal	Basal	4	0	8	5	5	23	8	5	5
Basal	Basal	5	0	27	43	43	—31	—39	1	0
Basal	Extra	1	0	20	17	17	—4	—9	0	0
Basal	Extra	2	0	18	23	23	—14	—24	1	0
Extra	Basal	11	—1	15	28	29	—9	—23	0	0
Extra	Basal	12	2	8	11	9	—9	—8	1	
Extra	Extra	8	0	46	56	56	—32	—44	3	
Extra	Extra	9	0	44	56	56	—28	—41	3	3
		<i>m</i>	0.1	23.2	29.9	29.8	—13.0	—22.5	1.8	
CALORIC GROUP T										
Basal	Basal	104	0	15	23	23	—16	—21	1	
Basal	Basal	105	0	42	30	30	—8	—20	1	
Basal	Extra	101	—3	23	47	50	—6	—21	3	
Basal	Extra	102	0	12	22	22	—5	—20		
Extra	Basal	111	0	26	25	25	—15	—24	1	
Extra	Basal	112	0	11	31	31	—15	—25	0	0
Extra	Extra	108	0	36	61	61	—50	—56	8	
Extra	Extra	109	0	19	27	27	—14	—24	2	2
		<i>m</i>	—0.4	23.0	33.2	33.6	—16.1	—26.4	2.3	
		<i>M</i>	—0.03	24.0	33.2	33.2	—14.0	—23.2		
		<i>SD</i>	0.69		15.2	15.4				

TABLE 510

DETERIORATION RATINGS. Each subject was rated by the same group of men at all periods, on a scale from 0 (normal) to 5 (extremely deteriorated). The value for a given subject is the average of these ratings.

Supplement		Subject	Period			
Protein	Vitamin		S12	S24	$\Delta R6$	$\Delta R12$
CALORIC GROUP Z						
Basal	Basal	122	1.2	1.5	—0.7	—1.0
Basal	Basal	123	0.7	2.6	—0.7	—1.4
Basal	Extra	119	0.7	1.8	—0.1	—1.0
Basal	Extra	120	1.8	1.7	—0.5	—1.1
Extra	Basal	129	2.0	2.1	—0.5	—1.2
Extra	Basal	130	2.7	3.7	—1.4	—2.3
Extra	Extra	126	0.9	2.1	—0.9	—1.3
Extra	Extra	127	1.8	1.8	—0.6	—1.1
		<i>m</i>	1.5	2.2	—0.7	—1.3
CALORIC GROUP L						
Basal	Basal	22	2.0	2.2	—1.0	—2.0
Basal	Basal	23	0.8	1.5	0.5	—0.7
Basal	Extra	19	1.3	1.7	—0.5	—1.0
Basal	Extra	20	1.2	3.0	—0.5	—1.7
Extra	Basal	29	3.7	3.1	—1.1	—1.6
Extra	Basal	30	1.6	2.4	—1.2	—2.0
Extra	Extra	26	1.5	2.1	—0.7	—1.7
Extra	Extra	27	1.5	3.2	—0.9	—1.7
		<i>m</i>	1.7	2.4	—0.7	—1.6
CALORIC GROUP G						
Basal	Basal	4	0.6	2.2	—0.9	—1.6
Basal	Basal	5	1.1	2.1	—0.9	—1.5
Basal	Extra	1	2.4	3.4	—2.2	—2.2
Basal	Extra	2	1.0	1.8	—0.8	—1.6
Extra	Basal	11	1.1	1.7	—1.3	—1.6
Extra	Basal	12	0.7	2.7	—0.9	—1.8
Extra	Extra	8	2.5	3.1	—1.7	—2.6
Extra	Extra	9	2.4	3.2	—1.7	—2.8
		<i>m</i>	1.5	2.5	—1.3	—2.0
CALORIC GROUP T						
Basal	Basal	104	1.5	2.5	—1.0	—1.5
Basal	Basal	105	1.3	1.8	—1.0	—1.5
Basal	Extra	101	1.7	3.0	—1.6	—2.2
Basal	Extra	102	1.2	1.9	—0.8	—1.5
Extra	Basal	111	2.1	2.1	—1.5	—1.9
Extra	Basal	112	0.9	1.9	—1.0	—1.8
Extra	Extra	108	3.0	3.6	—2.0	—3.2
Extra	Extra	109	0.9	1.1	—0.7	—1.1
		<i>m</i>	1.6	2.2	—1.2	—1.9
		<i>M</i>	1.6	2.3	—1.0	—1.7

TABLE 511

MINNESOTA MULTIPHASIC PERSONALITY INVENTORY, standard scores (normal average = 50, 1 *SD* = 10). Scales: Psychopathic Deviation (column 1), Paranoia (column 2), Psychasthenia (column 3), Schizophrenia (column 4), and Hypomania (column 5). All values are for the control period.

Supplement		Subject	1	2	3	4	5
Protein	Vitamin						
CALORIC GROUP Z							
Basal	Basal	122	46	44	49	52	42
Basal	Basal	123	50	54	39	40	48
Basal	Extra	119	54	62	40	44	46
Basal	Extra	120	54	60	57	54	40
Extra	Basal	129	52	56	49	46	50
Extra	Basal	130	58	48	76	79	60
Extra	Extra	126	31	53	44	44	42
Extra	Extra	127	53	54	39	41	58
		<i>m</i>	49.8	53.9	49.1	50.0	48.2
CALORIC GROUP L							
Basal	Basal	22	52	59	53	58	64
Basal	Basal	23	49	53	52	48	46
Basal	Extra	19	58	52	46	48	52
Basal	Extra	20	62	54	46	53	53
Extra	Basal	29	56	53	50	56	62
Extra	Basal	30	59	54	40	42	42
Extra	Extra	26	44	52	40	39	46
Extra	Extra	27	55	59	46	51	64
		<i>m</i>	54.4	54.5	46.6	49.4	53.6
CALORIC GROUP G							
Basal	Basal	4	48	58	37	40	53
Basal	Basal	5	63	64	40	46	48
Basal	Extra	1	46	56	48	48	48
Basal	Extra	2	60	52	44	46	54
Extra	Basal	11	52	56	46	44	44
Extra	Basal	12	68	59	40	43	60
Extra	Extra	8	54	52	38	42	52
Extra	Extra	9	48	54	50	48	54
		<i>m</i>	54.9	56.4	42.9	44.6	51.6
CALORIC GROUP T							
Basal	Basal	104	46	53	40	46	49
Basal	Basal	105	52	50	47	46	40
Basal	Extra	101	36	44	46	43	42
Basal	Extra	102	60	42	44	48	61
Extra	Basal	111	48	53	44	46	56
Extra	Basal	112	58	53	49	55	51
Extra	Extra	108	51	53	42	42	50
Extra	Extra	109	46	46	40	43	54
		<i>m</i>	49.6	49.2	44.0	46.1	50.4
		<i>M</i>	52.2	53.5	45.7	47.5	51.0
		<i>SD</i>	7.5	5.0	7.4	7.5	7.1

TABLE 512

MINNESOTA MULTIPHASIC PERSONALITY INVENTORY, standard scores (normal average = 50, 1 SD = 10). Scale: *Depression*.

Supplement			Period							
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20	R32
CALORIC GROUP Z										
Basal	Basal	122	54	76	74	20	15	10		56
Basal	Basal	123	52	63	70	18	—7	—10	75	63
Basal	Extra	119	55	64	71	16	—1	13	56	53
Basal	Extra	120	61	81	86	25	—11	4		
Extra	Basal	129	47	65	68	21	—10	—4	60	46
Extra	Basal	130	66	86	93	27	—13	—13		48
Extra	Extra	126	52	60	84	32	—7	—14		
Extra	Extra	127	45	54	68	23	—3	—4	51	51
		<i>m</i>	54.0	68.6	76.7	22.7	—4.6	—2.2		
CALORIC GROUP L										
Basal	Basal	22	54	60	64	10	—13	—17		46
Basal	Basal	23	63	66	72	9	—4	15	84	
Basal	Extra	19	52	66	56	4	0	—4		51
Basal	Extra	20	56	66	104	48	—5	—18		
Extra	Basal	29	49	69	83	34	—18	—24		51
Extra	Basal	30	57	68	80	23	0	—8		58
Extra	Extra	26	52	59	64	12	—20	—14	44	34
Extra	Extra	27	43	54	62	19	—6	0		60
		<i>m</i>	53.2	63.5	73.1	19.9	—8.2	—8.8		
CALORIC GROUP G										
Basal	Basal	4	49	59	80	31	2	—6	58	53
Basal	Basal	5	48	56	72	24	—7	—15		46
Basal	Extra	1	56	48	60	4	—9	—3		53
Basal	Extra	2	51	59	63	12	0	—6	53	48
Extra	Basal	11	67	72	78	11	—10	—3		65
Extra	Basal	12	54	60	57	3	11	1		
Extra	Extra	8	57	80	88	31	—18	—26		
Extra	Extra	9	58	78	95	37	—32	—20		51
		<i>m</i>	55.0	64.0	74.1	19.1	—7.9	—9.8		
CALORIC GROUP T										
Basal	Basal	104	52	68	76	24	—4	—20	51	
Basal	Basal	105	68	70	75	7	—10	—11		
Basal	Extra	101	58	66	72	14	—4	0	65	
Basal	Extra	102	45	52	52	7	—1	—4		
Extra	Basal	111	64	72	90	26	—22	—21		
Extra	Basal	112	54	68	70	16	7	—7	56	51
Extra	Extra	108	48	58	72	24	—14	—14		
Extra	Extra	109	49	50	65	16	—19	—20	39	36
		<i>m</i>	54.7	63.0	71.5	16.8	—8.4	—12.1		
		<i>M</i>	54.2	64.8	73.8	19.6	—7.3	—8.2		
		<i>SD</i>	6.5	9.2	12.1					

TABLE 513
MINNESOTA MULTIPHASIC PERSONALITY INVENTORY, standard scores (normal
average = 50, 1 SD = 10). Scale: *Hysteria*.

Supplement			Period							
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20	R32
CALORIC GROUP Z										
Basal	Basal	122	46	70	62	16	—2	5		40
Basal	Basal	123	47	54	60	13	—4	—12	49	51
Basal	Extra	119	43	50	51	8	—7	—3	40	40
Basal	Extra	120	43	61	64	21	—8	—8		
Extra	Basal	129	51	62	54	3	—1	5	53	47
Extra	Basal	130	51	70	72	21	—3	—6		53
Extra	Extra	126	44	46	62	18	—6	—6		
Extra	Extra	127	44	56	56	12	—7	—6	42	40
		<i>m</i>	46.1	58.6	60.1	14.0	—4.8	—3.9		
CALORIC GROUP L										
Basal	Basal	22	48	59	68	20	—10	—8		47
Basal	Basal	23	46	61	60	14	—13	6	49	
Basal	Extra	19	52	69	62	10	0	—13		40
Basal	Extra	20	44	58	82	38	—8	—10		
Extra	Basal	29	43	72	70	27	—3	—10		47
Extra	Basal	30	40	60	70	30	—3	—12		40
Extra	Extra	26	46	61	64	18	—15	—17	42	42
Extra	Extra	27	46	54	65	19	—14	—7		42
		<i>m</i>	45.6	61.8	67.6	22.0	—8.2	—8.9		
CALORIC GROUP G										
Basal	Basal	4	41	52	60	19	—9	—12	40	42
Basal	Basal	5	52	50	65	13	—12	—18		40
Basal	Extra	1	50	61	62	12	—4	—3		53
Basal	Extra	2	46	52	60	14	—9	—11	42	42
Extra	Basal	11	46	52	64	18	1	—10		49
Extra	Basal	12	46	50	58	12	—7	—12		
Extra	Extra	8	43	66	70	27	—19	—19		
Extra	Extra	9	51	72	70	19	—17	—14		40
		<i>m</i>	46.9	56.9	63.6	16.7	—9.5	—12.4		
CALORIC GROUP T										
Basal	Basal	104	46	56	51	5	2	—9	40	
Basal	Basal	105	50	62	64	14	—15	—5		
Basal	Extra	101	43	52	62	19	—2	—8	42	
Basal	Extra	102	46	53	56	10	—7	—7		
Extra	Basal	111	43	60	72	29	—14	—21		
Extra	Basal	112	41	53	62	21	—4	—12	40	42
Extra	Extra	108	41	53	56	15	—3	—8		
Extra	Extra	109	44	54	62	18	—9	—14	44	42
		<i>m</i>	44.2	55.4	60.6	16.4	—6.5	—10.5		
		<i>M</i>	45.7	58.2	63.0	17.3	—7.2	—8.9		
		<i>SD</i>	3.4	7.1	6.6	7.4	5.5	6.4		

TABLE 514

MINNESOTA MULTIPHASIC PERSONALITY INVENTORY, standard scores (normal average = 50, 1 SD = 10). Scale: *Hypochondriasis*.

Supplement			Period							
Protein	Vitamin	Subject	C	S12	S24	dS24	△R6	△R12	R20	R32
CALORIC GROUP Z										
Basal	Basal	122	42	56	52	10	4	6		38
Basal	Basal	123	63	70	78	15	—3	—7	73	75
Basal	Extra	119	68	74	76	8	—3	—6	69	65
Basal	Extra	120	57	54	60	3	2	2		
Extra	Basal	129	61	70	73	12	—6	—2	76	65
Extra	Basal	130	44	59	68	24	—8	—9		53
Extra	Extra	126	54	60	69	15	6	—5		
Extra	Extra	127	61	70	68	7	—1	—5	64	60
		<i>m</i>	56.2	64.1	68.0	11.8	—1.1	—3.2		
CALORIC GROUP L										
Basal	Basal	22	54	58	60	6	—9	—6		51
Basal	Basal	23	52	62	69	17	—7	2	69	
Basal	Extra	19	64	71	66	2	5	—2		53
Basal	Extra	20	53	64	88	35	—1	—4		
Extra	Basal	29	58	78	70	12	1	3		55
Extra	Basal	30	64	78	86	22	—4	—14		69
Extra	Extra	26	61	68	74	13	—10	—13	60	60
Extra	Extra	27	65	68	76	11	—3	—2		60
		<i>m</i>	58.9	68.4	73.6	14.7	—3.5	—4.5		
CALORIC GROUP C										
Basal	Basal	4	66	66	76	10	0	—5	64	64
Basal	Basal	5	64	67	78	14	—5	—11		62
Basal	Extra	1	60	67	72	12	—7	—4		64
Basal	Extra	2	62	66	74	12	—7	—6	62	62
Extra	Basal	11	62	70	76	14	0	—6		67
Extra	Basal	12	62	60	64	2	1	—2		
Extra	Extra	8	59	64	64	5	—9	—5		
Extra	Extra	9	70	78	76	6	—5	—10		56
		<i>m</i>	63.1	67.2	72.5	9.4	—4.0	—6.1		
CALORIC GROUP T										
Basal	Basal	104	58	67	54	—4	11	1	56	
Basal	Basal	105	58	68	68	10	—12	—6		
Basal	Extra	101	50	45	56	6	—7	—7	40	
Basal	Extra	102	58	59	64	6	—8	—5		
Extra	Basal	111	59	65	74	15	—12	—11		
Extra	Basal	112	57	70	68	11	—1	—9	56	56
Extra	Extra	108	61	67	74	13	—7	—6		
Extra	Extra	109	60	66	69	9	—7	—9	58	58
		<i>m</i>	57.6	63.4	65.9	8.3	—5.4	—6.5		
		<i>M</i>	59.0	65.8	70.0	11.0	—3.5	—5.1		
		<i>SD</i>	6.1	7.1	8.2	7.2	5.4	4.6		

TABLE 515

SEMEN COLLECTION, RECORD OF RESPONSE. S = sample was obtained; T = no sample could be secured; N = did not participate in collection. Blank spaces indicate that no record is available for the subject. At the end of semi-starvation (S24), the semen collection was made on two occasions, five days apart (S24A and S24B).

Supplement		Subject	Period			
Protein	Vitamin		S24A	S24B	R7	R10
CALORIC GROUP Z						
Basal	Basal	122	T		T	T
Basal	Basal	123	T		S	S
Basal	Extra	119	T		T	T
Basal	Extra	120	S	S	N	S
Extra	Basal	129	S		S	N
Extra	Basal	130	N	N	N	N
Extra	Extra	126	S	S	S	S
Extra	Extra	127	S	S	S	S
CALORIC GROUP L						
Basal	Basal	22	T		N	
Basal	Basal	23		S	T	S
Basal	Extra	19	T		T	T
Basal	Extra	20	S	S	S	S
Extra	Basal	29	T	T	T	
Extra	Basal	30	S	S	S	S
Extra	Extra	26	S	S	S	S
Extra	Extra	27	S	S	S	S
CALORIC GROUP G						
Basal	Basal	4	T		N	
Basal	Basal	5	S	S	S	S
Basal	Extra	1	T		T	T
Basal	Extra	2	N	N	N	N
Extra	Basal	11	S	S	S	S
Extra	Basal	12	T		N	N
Extra	Extra	8	S	S	S	S
Extra	Extra	9	T		N	T
CALORIC GROUP T						
Basal	Basal	104	T		T	
Basal	Basal	105	N	N	N	N
Basal	Extra	101	S	S	T	S
Basal	Extra	102	N	S	S	S
Extra	Basal	111	S	S	S	S
Extra	Basal	112	T		T	T
Extra	Extra	108	T		T	T
Extra	Extra	109	T	S	S	S

TABLE 516

PHYSICAL CHARACTERISTICS OF SPERM AND SEMEN. Den. = density, number of sperm per cc. of semen. Viab. = viability, per cent showing motility at original observation. Orig. Mot. = original motility. Visc. = viscosity, graded on an arbitrary scale where normal semen is 1. Turb. = turbidity.

Subject	Vol- ume (cc.)	Visc.	Turb.	pH	Count $\times 10^6$		Viab.	Orig. Mot.	Life (hrs.)
					Den.	Total			
S24A									
5	1.7	1+	3+	8	120	204	90	1+	<8
8	0.1	2+	2+	6	39	3.9	70	1+	<8
11	5.0	2+	3+	7	165	825	40	3+	<7
20	0.5	3+	4+	7	1500	750	100	0	0
26	1.1	2+	4+	7	430	473	10	3+	<8
27	0.2	3+	3+	7	367	73	80	2+	<8
30	0.3	3+	4+	7	1470	441	30	3+	8
101	0.2	2+	2+	6	0	0	100	0	0
111	0.1	1+	1+	8	25	2.5	40	2+	<8
120	2.0	2+	3+	8	355	710	80	2+	7
126	3.0	1+	2+	8	120	360	70	2+	7
127	0.5	2+	3+	7	96	48	80	1+	8
129	0.4	2+	3+	7	745	335	20	3+	<17
S24B									
5	1.0	2+	2+	8	105	105	70	2+	6
8	0.05	2+	1+	6	2ns		100		0
11	5.6	1+	1+	7	75	420	70	1+	8
20	0.2	2+	3+	8	180	36	100	0	0
23	3.5	2+	4+	7	340	1190	60	2+	4
26	0.9	2+	1+	8	730	657	80	1+	7
27	0.4	4+	3+	8	1410	564	95	1+	<6
30	0.3	3+	4+	7	2710	813	60	2+	6
101	0.4	2+	4+	7	630	220	70	2+	5
102	3.0	2+	4+	7	1230	3660	70	2+	7
109	3.5	2+	4+	8	730	2555	70	2+	8
111	2.0	1+	1+	8	25	5	70	1+	7
120	1.4	2+	3+	8	200	280	50	2+	7
126	2.5	2+	4+	7	90	225	90	1+	3
127	0.1	2+	2+	8	195	20	90	1+	6
R7									
5	1.3	1+	1+		130	169	50	2+	<18
8	0.3	2+	3+	7	160	48	20		24
11	6.5	3+	3+	7	230	1495	80	1+	24
20	0.4	1+	1+	8	16	6	100	0	0
26	1.8	2+	3+	7	320	576	25	2+	24
27	0.2	2+	2+	8	750	150	100	0	0
30	0.2	3+	3+	7	1780	267	90	2+	<23
102	4.5	3+	3+	7	795	3578	60	2+	20
109	4.0	3+	3+	7	340	1360	50	3+	18
111	0.4	2+	3+	7	105	37	70	2+	6
123	0.9	3+	3+	8	780	702	85	1+	14
126	2.3	2+	3+	7	72	166	50	3+	<20
127	0.7	2+	2+	7	74	52	80	2+	11
129	0.9	3+	3+	7	770	693	20	3	24

TABLE 516 *continued*

Subject	Volume (cc.)	Visc.	Turb.	pH	Count $\times 10^6$		Viab.	Orig. Mot.	Life (hrs.)
					Den.	Total			
R10									
5	3.0	2+	2+		18	52	60	2+	13
8	0.4	2+	3+	8	495	198	50	2+	24
11	4.0	2+	2+		28	109	50	2+	23
20	1.1	2+	1+	8	2	2	100	0	0
23	3.5	2+	3+	7	292	1024	60	2+	24
26	1.5	2+	2+		100	150	50	3+	<20
27		3+	1+		80		95	\pm	<8
30	0.9	3+	3+	7	255	230	50	2+	<21
101	1.3	1+	3+	7	175	228	10	3+	23
102	5.5	2+	3+	7	130	715	75	2+	23
109	5.0	1+	3+	8	62	312	40	2+	24
111		2+	2+	7	65				
120	2.1	2+	3+	8	500	1050	90	1+	15
123	1.8	3+	4+		230	414	90	1+	<15
126	2.2	2+	2+	7	20	44	85	2+	13
127	0.5	2+	2+		158	79	75	1+	<24
R20									
5	2.5	2+	2+	7	1	2	10	3+	32
23	4.5	3+	2+	7	30	135	20	3+	36
26	6.0	3+	3+	7	25	150	16	3+	35
101	2.3	3+	2+	7	70	161	0	3+	34
109	4.5	3+	2+	8	70	315	40	2+	32
123	4.3	2+	3+	7	25	108	80	2+	15
127	4.5	1+	2+	7	2	9	85	2+	30
129	5.0	3+	3+	7	105	525	10	3+	31
R58									
23	7.0	3+	3+		35	245	60	3+	24
101	3.5	1+	2+		60	210	80	3+	<24
102	9.5	2+	3+		200	1900	90	3+	24
109	7.0	2+	2+		150	1050	90	4+	24

TABLE 517

SPERM, HISTOLOGICAL EXAMINATION. A semen smear stained with gentian violet was used in each case. In the first section are given the actual number of sperm examined and the percentages of those which were either normal or abnormal in any way. The other three sections indicate the breakdown of the abnormalities. The separate abnormalities are given as per cent of all cells examined. Abn. = abnormal shape of head, neck, or tail as per section. Bent = neck bent abnormally. Coil = coiled tail. Dbl. = double head or tail as per section. Lg. = head excessively large. Short = tail excessively short. Sm. = head excessively small.

Subject	Sperm Examined			Head					
	No.	% Norm.	% Abn.	Lg.	Sm.	Dbl.	Abn.	Misc.	Total
S24A									
5	115	84.3	15.7		6.2		0.9	1.7	8.8
8	200	65.5	34.5		8.0		8.0		16.0
11	265	71.7	28.3		4.1		4.5		8.6
20	204	66.2	33.8		6.1	0.4	6.0		12.5
26	109	85.3	14.7		0.9		2.8	0.9	4.6
27	200	71.5	28.5	0.5	6.0	0.5	3.0	1.0	11.0
30	210	79.5	20.5		5.7		0.5		6.2
111	200	51.0	49.0	1.5	10.0	1.0	6.0	6.5	25.0
120	206	54.9	45.1	1.0	15.0		2.9	2.9	21.8
126	229	49.8	50.2		10.9	0.4	19.7		31.0
127	200	68.0	32.0	3.0	9.0		2.0	3.0	17.0
129	222	56.3	43.7	0.5	9.3	0.5	5.4		15.7
S24B									
5	200	71.5	28.5	1.0	6.0		5.0		12.0
11	218	72.0	28.0	0.5	4.5	0.5	6.9	0.5	12.9
20	223	71.7	28.3		6.7	1.3	4.5		12.5
23	205	69.2	30.8	1.5	2.3		5.4	1.0	10.2
26	200	70.0	30.0	0.5	8.5		6.5	0.5	16.0
27	200	60.5	39.5	4.0	12.5		9.0		25.5
30	200	76.8	23.2	0.5	5.0		4.0		9.5
101	207	51.7	48.3	0.5	8.2		3.9		12.6
102	215	53.5	46.5	0.5	2.4	0.5	6.5		9.9
109	213	68.1	31.9		0.9		2.8		3.7
111	200	55.0	45.0	1.5	7.0	0.5	5.0	3.5	17.5
120	197	64.0	36.0		5.7		3.0	1.5	10.2
126	234	57.7	42.3	1.3	9.3	0.9	12.0		23.5
127	200	69.0	31.0	3.0	6.0		3.5		12.5
R7									
5	109	75.2	24.8		5.5	0.9	2.8		9.2
8	109	82.6	17.4		2.7				2.7
11	205	66.3	33.7		7.8		2.9		10.7
20	210	81.0	19.0		9.0		1.9		10.9
26	210	69.0	31.0	0.5	6.1		2.9		9.5
27	105	81.9	18.1	1.9	1.8				3.7
30	213	73.7	26.3	0.5	6.5	0.5	0.5		8.0
102	104	76.0	24.0		4.7				4.7
109	112	71.4	28.6	0.9	2.6		5.4		8.9
111	111	72.1	27.9	2.7	5.4		3.6		11.7
123	132	68.9	31.1		9.1		6.1		15.2
126	125	60.8	39.2	3.2	20.0		0.8		24.0
127	100	78.0	22.0		4.0		4.0		8.0
129	111	56.8	43.2	0.9	23.4				24.3

TABLE 517 *continued*

Subject	Sperm Examined			Head					
	No.	% Norm.	% Abn.	Lg.	Sm.	Dbl.	Abn.	Misc.	Total
R10									
5	100	67.0	33.0	1.0	13.0				14.0
8	200	76.5	23.5		13.5		0.5		14.0
11	100	79.0	21.0		4.0		3.0		7.0
20	105	72.4	27.6	5.7	3.8		3.8	2.9	16.2
23	100	82.0	18.0		2.0		2.0		4.0
26	100	72.0	28.0		10.0		1.0		11.0
27	106	74.5	25.5		8.5		2.8		11.3
30	200	80.5	19.5	1.0	4.8		0.5		6.3
101	200	88.5	11.5	0.5	4.0		0.5		5.0
102	200	79.5	20.5	1.0	4.5		2.5		8.0
109	200	88.0	12.0	0.5	3.0	0.5			4.0
111	200	70.5	29.5	1.5	5.5		1.5		8.5
120	200	81.5	18.5	1.0	3.5		0.5		5.0
123	132	68.9	31.1		9.1		6.1		15.2
126	200	77.5	22.5	2.0	8.5		1.5		12.0
127	100	78.0	22.0		4.0		4.0		8.0
R20									
23	281	64.1	35.9		3.4	0.4	4.3	0.4	8.5
26	200	86.5	13.5	0.5	5.5		1.5		7.5
101	205	78.5	21.5	0.5	4.3		2.0	1.5	8.3
104	100	58.0	42.0		6.0		2.0		8.0
109	105	63.8	36.2	1.0	5.7		5.7	3.8	16.5
123	200	80.5	19.5	0.5	6.5		4.0		11.0
127	100	79.0	21.0	3.0	3.0		5.0		11.0
129	206	55.8	44.2	0.5	8.2		5.3	1.5	15.5
S24A									
5	0.9		0.9	4.3				1.7	6.0
8	4.5	1.5	6.0	9.5	0.5		0.5	2.0	12.5
11	7.5	0.8	8.3	10.6	0.4		0.4		11.4
20	2.5	4.9	7.4	9.4	2.0			2.5	13.9
26	5.5	0.9	6.4	3.7					3.7
27	4.5	0.5	5.0	10.5	1.0			1.0	12.5
30	1.9		1.9	11.9				0.5	12.4
111	8.0	5.0	13.0	8.0	0.5		1.0	1.5	11.0
120	4.4	2.4	6.8	16.5					16.5
126	4.4	1.3	5.7	9.6	2.2		0.4	1.3	13.5
127	0.5	0.5	1.0	12.0	1.0		1.0		14.0
129	5.0	8.1	13.1	14.4	0.5				14.9
S24B									
5	1.5	2.0	3.5	12.5	0.5				13.0
11	5.0		5.0	8.7	0.9			0.5	10.1
20	1.3	4.5	5.8	9.0	0.5			0.5	10.0
23	1.5	2.0	3.5	15.6				1.5	17.1
26	2.5	2.0	4.5	9.5					9.5
27	3.5	0.5	4.0	8.5	1.5				10.0
30		0.5	0.5	13.2					13.2

TABLE 517 *continued*

Subject	Neck			Tail				
	Bent	Abn.	Total	Coil	Dbl.	Short	Abn.	Total
101	2.9	2.4	5.3	27.5	1.0	0.5	1.4	30.4
102	5.1	0.9	6.0	26.5	2.3	0.9	0.9	30.6
109	3.8	5.6	9.4	17.4	0.9		0.5	18.8
111	11.0	3.5	14.5	10.5	1.5	1.0		13.0
120	0.5	2.0	2.5	21.3	0.5		1.5	23.3
126	3.8		3.8	12.0	3.0			15.0
127	2.0		2.0	15.0	0.5		1.0	16.5
R7								
5	4.6		4.6	10.1	0.9			11.0
8		0.9	0.9	13.8				13.8
11	5.9	1.0	6.9	15.1	1.0			16.1
20	4.3	0.9	5.2	2.4		0.5		2.9
26	4.8	2.4	7.2	13.8	0.5			14.3
27	2.9	2.9	5.8	7.6	1.0			8.6
30	5.2		5.2	13.1				13.1
102	1.9	2.9	4.8	13.5		1.0		14.5
109	4.5	3.6	8.1	11.6				11.6
111	1.8		1.8	13.5	0.9			14.4
123	5.3	0.8	6.1	8.3	1.5			9.8
126	3.2		3.2	8.0	4.0			12.0
127	8.0	1.0	9.0	5.0				5.0
129	1.8	0.9	2.7	16.2				16.2
R10								
5	4.0	1.0	5.0	14.0				14.0
8	2.0		2.0	5.5	0.5		1.5	7.5
11	7.0	1.0	8.0	3.0	1.0	1.0	1.0	6.0
20	1.9	5.7	7.6		3.8			3.8
23	1.0		1.0	12.0	1.0			13.0
26	4.0	2.0	6.0	9.0	1.0	1.0		11.0
27	1.9	1.0	2.9	9.4	1.9			11.3
30	4.5	0.5	5.0	8.2				8.2
101	0.5	2.5	3.0	3.5				3.5
102	2.5		2.5	9.5	0.5			10.0
109	2.5	0.5	3.0	4.5		0.5		5.0
111	2.5	0.5	3.0	17.5		0.5		18.0
120	5.0	0.5	5.5	7.0		0.5	0.5	8.0
123	5.3	0.8	6.1	8.3	1.5			9.8
126	2.0	0.5	2.5	7.0	1.0			8.0
127	8.0	1.0	9.0	5.0				5.0
R20								
23	1.1	2.5	3.6	22.0	0.4	0.7	0.7	23.8
26	1.5		1.5	3.5	0.5		0.5	4.5
101	1.5	3.4	4.9	7.3	0.5	0.5		8.3
104	7.0	1.0	8.0	26.0				26.0
109	1.9	1.9	3.8	15.2	1.0			16.2
123	1.0	1.0	2.0	5.5	0.5		0.5	6.5
127	6.0		6.0	4.0				4.0
129	4.4	3.9	8.3	19.4	0.5	0.5		20.4

TABLE 518

ELECTROPHORETIC ANALYSIS of blood serum from 2 men before semi-starvation (control), after 12 and 24 weeks of semi-starvation (S12 and S24), and after 6, 12, 20, and 34 weeks of rehabilitation (R6, R12, R20, and R34). Values in gm. per 100 cc. for total serum proteins, and as per cent of total proteins represented by the several fractions.

Period	Total	A/G	% Albumin	% Alpha ₁	% Alpha ₂	% Beta	% Gamma
Subject 122							
Control	6.89	1.70	62.8	4.9	8.4	11.8	12.1
S12	7.10	2.30	69.6	3.5	7.7	10.5	8.7
S24	6.88	1.70	63.0	4.7	9.7	12.9	9.7
R6	6.78	2.33	70.0	3.3	7.1	11.6	8.0
R12	7.10	1.95	66.2	4.2	8.0	13.0	8.6
R34	6.18	1.65	62.2	4.3	8.4	15.2	9.9
Subject 123							
Control	6.99	1.76	63.7	4.4	7.6	11.2	13.1
S12	7.18	2.40	70.6	3.2	6.4	9.9	9.9
S24	6.16	1.88	65.4	3.2	7.5	13.7	10.2
R6	6.43	2.00	66.7	3.3	7.0	13.2	9.8
R12	7.24	1.91	65.5	3.3	7.2	13.1	10.9
R20	6.20	1.47	59.5	4.4	9.8	14.0	12.3
R34	6.60	1.58	61.2	4.1	9.2	15.0	10.5

Tables of Diets

TABLE 519

COMPOSITION OF REPRESENTATIVE MENUS SERVED DURING THE CONTROL PERIOD. All values have been calculated from standard tables of food composition. These are the diets listed in Table 520.

	Protein			Fat			Carbohydrate			Total
	Gm.	Cal.	% Total	Gm.	Cal.	% Total	Gm.	Cal.	% Total	
January 25	96.2	384.8	12.2	130.6	1175.4	37.4	396.5	1586.0	50.4	3146.2
January 30	105.7	422.8	13.0	142.6	1283.4	39.3	389.2	1556.8	47.7	3263.0
February 4	97.7	390.8	12.6	129.4	1164.6	37.5	386.7	1546.8	49.9	3102.2
February 8	103.0	412.0	12.8	121.5	1093.5	34.1	426.2	1704.8	53.1	3210.3

TABLE 520

REPRESENTATIVE DIETS SERVED DURING THE CONTROL PERIOD

Food Served	Gm. Weight of Portion	Food Served	Gm. Weight of Portion	Food Served	Gm. Weight of Portion
January 25, 1945					
Grapefruit juice	100	Rice, steamed	100	Spice cake, iced ...	60
Corn flakes	30	Lamb, fricasseed ...	50	Roast veal	50
Bread, white	120	Gravy	120	Potatoes, whipped ..	210
Butter	30	Peas, steamed	100	Corn	100
Milk	720	Carrot, raisin salad .	75	Waldorf salad	75
Jam	70	Mayonnaise	15	Ice cream	60
Sugar	10	Bran muffins	70	Cooky	20
Tomato juice	75				
January 30, 1945					
Grapefruit juice	100	Crackers, soda	8	Beef roast	60
Bran flakes	30	Macaroni and		Gravy	75
Bread, white	180	cheese	200	Parsnips	100
Butter	30	Eggs, scrambled ...	60	Potatoes, whipped .	200
Milk	720	Bacon	20	Tomato salad	75
Jam	70	Cabbage salad	75	Mayonnaise	10
Sugar	10	Sponge cake	30	Ice cream	75
Vegetable soup	110	Orange sauce	30	Wafer	5
February 4, 1945					
Tomato juice	100	Apple juice	75	Liverwurst	35
Corn flakes	30	Pork roast	60	Egg salad	35
Bread	180	Gravy	75	Carrot sticks	35
Jam	35	Potatoes, whipped .	200	Celery sticks	35
Sugar	10	Peas, steamed	100	Olives	20
Butter	30	Fruit salad	110	Chocolate cake,	
Milk	720	Lettuce	20	iced	60
Sweet roll	50	Chocolate sundae ..	78	Apple	150
February 8, 1945					
Vegetable juice ...	100	Creamed corn soup.	110	Lettuce	15
Bran flakes	30	Crackers, soda	12	Apple Betty	100
Jam	70	Meat loaf	60	Pork chops	60
Bread	180	Gravy	75	Sweet potatoes	170
Sugar	10	Potatoes, whipped..	200	Peas	100
Butter	30	Green beans	100	Applesauce	100
Milk	720	Apricots	45	Ginger cake	60

TABLE 521

VITAMIN CONTENT OF THE DIETS SERVED DURING THE CONTROL PERIOD. Values are given for three weeks of the control period. Each figure is the mean of the diets served during that week. All values have been calculated from standard tables of food composition.

Period	Vita- min A	Thiamine	Ribo- flavin	Niacin	Vita- min C
C7	8412	1.86	2.49	16.0	104
C8	8754	1.99	2.54	14.5	104
C9	8498	1.74	2.52	15.6	117
Average	8555	1.86	2.52	15.4	108

TABLE 522

COMPOSITION OF BASAL DIET I IN THE SEMI-STARVATION PERIOD. All values have been calculated from standard tables of food composition.

	Weight	Protein	Fat	CHO	Calories
Cereals					
Bread	180	20.9	6.5	86.1	486.5
Farina	40	4.6	0.4	30.4	143.6
Flour	2	0.2		1.5	6.8
Oatmeal	7	1.0	0.5	5.1	28.9
Spaghetti	35	4.6	0.5	25.9	126.5
Dairy products					
Fats	17		15.7		141.3
Milk	60	2.1	2.3	2.9	40.7
Fruits and vegetables					
Apples	40	0.1	0.1	6.0	25.3
Cabbage	100	1.4	0.2	5.3	28.6
Carrots	17	0.2		1.6	7.2
Celery	11	0.1		0.4	2.0
Onions	16	0.2		1.6	7.2
Peas	38	1.4	0.2	5.2	28.2
Potatoes	350	7.0	0.4	66.5	297.6
Tomatoes	60	0.6	0.1	2.3	12.5
Meat and fish					
Beef	20	3.9	2.6		39.0
Fish	7	1.3	0.2		7.0
Sweets					
Jam	20	0.1		14.2	57.2
Jello	12	1.1		10.6	46.8
Sugar	20			20.0	80.0
Total	1052	50.8	29.7	285.6	1612.9

TABLE 523

COMPOSITION OF BASAL DIET II IN THE SEMI-STARVATION PERIOD. All values have been calculated from standard tables of food composition.

	Weight	Protein	Fat	CHO	Calories
Cereals					
Bread	180	20.9	6.5	86.1	486.5
Gingerbread	50	2.1	6.2	31.1	188.6
Flour	2	0.2		1.5	6.8
Oatmeal	30	4.3	2.2	20.5	119.0
Macaroni	50	6.5	0.7	36.9	179.9
Dairy products					
Cheese	35	1.2	1.1	0.1	15.1
Fats	17		17.0		153.0
Milk	140	4.9	5.5	6.9	96.7
Fruits and vegetables					
Lettuce	80	1.0	0.2	2.3	15.0
Rutabagas	40	0.4		3.6	16.0
Dried beans	16	2.6	0.2	9.4	49.8
Yellow split peas..	5	1.2		3.1	17.2
Potatoes	300	6.0	0.3	57.0	254.7
Meat and fish					
Ham	15	2.3	4.6		50.6
Sweets					
Jam	20	0.1		14.2	57.2
Sugar	20			20.0	80.0
Total	1000	53.7	44.5	292.7	1786.1

TABLE 524

COMPOSITION OF BASAL DIET III IN THE SEMI-STARVATION PERIOD. All values have been calculated from standard tables of food composition.

	Weight	Protein	Fat	CHO	Calories
Cereals					
Bread	185	21.5	6.7	88.5	500.3
Cornbread	110	12.7	9.2	45.4	315.2
Pancakes	100	7.4	3.5	37.0	209.1
Dairy products					
Fats	5		5.0		45.0
Milk	30	1.0	1.1	1.5	19.9
Fruits and vegetables					
Applesauce	70	0.2	0.1	13.8	56.9
Green beans	35	0.4		1.3	6.8
Carrots	20	0.2		1.6	7.2
Turnips	90	1.0	0.2	6.4	31.4
Onions	20	0.3		2.1	9.6
Potatoes	304	6.1	0.3	57.8	258.3
Meat and fish					
Beef	25	4.8	3.2		48.0
Sweets					
Jam	20	0.1		14.2	57.2
Syrup	50			37.0	148.0
Total	1064	55.1	29.3	306.6	1712.9
Total average caloric composition of the 3 diets.....					1704

TABLE 525

VITAMIN AND MINERAL CONTENT OF THE BASAL DIETS IN THE SEMI-STARVATION PERIOD. The values for thiamine, calcium, chloride, and phosphorus were determined on these diets by actual analysis whereas the others were calculated from standard tables of food composition.

Diet	Vitamins					Minerals			
	Vit. A (I.U.)	Thiamine (mg.)	Ribo- flavin (mg.)	Niacin (mg.)	C (mg.)	Fe (mg.)	Ca (gm.)	Cl as NaCl (gm.)	P (gm.)
I	1559	1.14	0.481	22.7	119	11.4	0.59	13.88	1.01
II	1964	1.56	0.730	19.5	53	12.6	0.97	13.46	1.27
III	1277	1.17	0.604	20.0	77	12.6	0.73	9.03	1.44
Average daily intake	1600	1.29	0.605	20.7	83	12.2	0.76	12.12	1.24

TABLE 526

COMPOSITION OF RELIEF MEAL served on May 26, 1945, the mid-point of the semi-starvation period in the Minnesota Experiment. The values for the composition of the diets were secured from standard tables of food composition.

	Wt.	Cal.		Wt.	Cal.		Wt.	Cal.
Grapefruit			Fruit punch ...	100	53	Strawberries ..	90	50
juice	100	67	Chicken	70	87	Baking-powder		
Bacon	10	67	Dressing	70	125	biscuits	70	194
Egg	50	79	Potatoes	150	244	Celery	10	2
Bread	105	278	Gravy	60	73	Peanut butter ..	13	89
Butter	15	111	Corn	90	87	Minced ham ..	23	60
Honey	15	48	Carrot salad ..	80	32	Jelly roll	30	160
Milk	600	410				Orange	100	50

Composition: Protein, 88.0 gm. Fat, 95.0 gm. CHO, 289.7 gm. Total calories, 2366.
% of total calories: Protein, 14.9. Fat, 36.1. CHO, 49.0.

COMPOSITION OF THE BASIC DIETS IN THE REHABILITATION PERIOD FOR THE DIFFERENT CALORIC GROUPS. The moisture, protein (Kjeldahl), fat, ash, and thiamine (thiochrome) contents of these diets were determined by actual analysis; the vitamin A, riboflavin, niacin, and ascorbic acid contents were calculated from standard tables of food composition. The carbohydrate and protein totals were secured by subtracting the sum of the moisture, fat, and ash figures from the weight of food served. The factor used in the conversion of carbohydrates and proteins to calories was 4, that for fat was 9. The three

separate diets were served in rotation on successive days. The vitamin contents are given in mg. per serving except in the case of vitamin A where it is expressed as I.U. per serving. These values do not coincide precisely with the averages of actual individual intakes because the latter were adjusted according to estimated balance requirements. Abbreviations: wh. = whole wheat bread; cas. = bread = casein bread; sw. pot. = sweet potatoes; muf. = senamine muffins; muf. van. = vanilla muffins; har. = beets; car. salad = carrot salad.

DIET I. HIGH PROTEIN GROUPS, July 30–September 9, 1945

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing Gm.		Vitamin Content per Serving				
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C		
CALORIC GROUP Z																		
Basic I	1400	78.7	1102	2.3	32.2	2.2	30.8	249	1.3	18.2	91	1273	2818	1.19	2.65	4.25	129	
Salad A	280	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Soy bread	180	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total	1860		1407		74.4		35.1	389		28.1		1873	3795	1.58	3.07	10.78	142	
CALORIC GROUP L																		
Basic I	1765	78.7	1389	2.3	40.6	2.2	38.8	314	1.3	22.9	91	1606	3552	1.50	3.34	5.39	163	
Salad A	280	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Soy bread	180	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total	2225		1694		82.8		43.1	454		32.8		2206	4529	1.89	3.76	11.89	176	
CALORIC GROUP G																		
Basic I	2130	78.7	1676	2.3	49.0	2.2	46.9	379	1.3	27.7	91	1940	4289	1.81	4.04	6.51	196	
Salad A	280	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Soy bread	180	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total	2590		1981		91.2		51.2	519		37.6		2540	5266	2.20	4.46	13.01	209	
CALORIC GROUP T																		
Basic I	2525	78.7	1987	2.3	58.1	2.2	55.6	449	1.3	32.8	91	2299	5080	2.14	4.78	7.72	232	
Salad A	280	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Soy bread	180	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total	2985		2292		100.3		59.9	589		42.7		2899	6057	2.52	5.20	14.22	245	

TABLE 528

DIET I. LOW PROTEIN GROUPS, July 30-September 9, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Gm.		Vitamin Content per Serving			
	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
CALORIC GROUP Z																
Basic I	78.7	1102	2.3	32.2	2.2	30.8	249	1.3	18.2	91	1273	2818	1.19	2.65	4.28	129
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.48	23
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40	
Total		1430		63.3		37.4	387		25.9		1886	3719	1.63	2.97	11.16	152
CALORIC GROUP L																
Basic I	78.7	1389	2.3	40.6	2.2	38.8	314	1.3	22.9	91	1606	3552	1.50	3.34	5.39	163
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.48	23
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40	
Total		1717		71.7		45.4	452		30.6		2219	4453	1.94	3.66	12.27	186
CALORIC GROUP G																
Basic I	78.7	1676	2.3	49.0	2.2	46.9	379	1.3	27.7	91	1940	4289	1.81	4.04	6.51	196
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.48	23
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40	
Total		2004		80.1		53.5	517		35.4		2553	5190	2.25	4.36	13.39	219
CALORIC GROUP T																
Basic I	78.7	1987	2.3	58.1	2.2	55.6	449	1.3	32.8	91	2299	5080	2.14	4.78	7.72	232
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.48	23
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40	
Total		2315		89.2		62.2	587		40.5		2912	5981	2.58	5.10	14.60	255

DIET I. HIGH PROTEIN GROUPS, September 10-October 4, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing		Vitamin Content per Serving				
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Vit. A	Thia- mine	Fla- vin	Nic- cin	Vit. C
CALORIC GROUP Z																		
Basic I	1400	78.7	1102	2.3	32.2	2.2	30.8	2.2	249	1.3	18.2	91	1273	2818	1.19	2.65	4.28	129
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	2.8	212	2.3	7.6	282	931		0.14	0.52	8.91	
Salad A	280	86.9	243	4.8	13.6	0.1		0.1	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.1	1.4	155	194	4393	0.09	0.09	1.24	21
Butter	20	16.0	3			80.5	16.1			2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		3	0.01	0.04		
Total	2187		1539		105.8		59.7		558		31.2		2773	8791	1.54	3.41	16.13	163
CALORIC GROUP L																		
Basic I	1765	78.7	1389	2.3	40.6	2.2	38.8	2.2	314	1.3	22.9	91	1606	3552	1.50	3.34	5.39	163
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	2.8	212	2.3	7.6	282	931		0.14	0.52	8.91	
Salad A	280	86.9	243	4.8	13.6	0.1		0.1	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.1	1.4	155	194	4393	0.09	0.09	1.24	21
Butter	20	16.0	3			80.5	16.1			2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		3	0.01	0.04		
Total	2552		1826		114.2		67.7		623		35.9		3106	9525	1.85	4.10	17.24	197
CALORIC GROUP G																		
Basic I	2130	78.7	1676	2.3	49.0	2.2	46.9	2.2	379	1.3	27.7	91	1940	4289	1.81	4.04	6.51	196
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	2.8	212	2.3	7.6	282	931		0.14	0.52	8.91	
Salad A	280	86.9	243	4.8	13.6	0.1		0.1	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.1	1.4	155	194	4393	0.09	0.09	1.24	21
Butter	20	16.0	3			80.5	16.1			2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		3	0.01	0.04		
Total	2917		2113		122.6		75.8		688		40.7		3440	10262	2.16	4.80	18.36	230
CALORIC GROUP T																		
Basic I	2525	78.7	1987	2.3	58.1	2.2	55.6	2.2	449	1.3	32.8	91	2299	5080	2.14	4.78	7.72	232
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	2.8	212	2.3	7.6	282	931		0.14	0.52	8.91	
Salad A	280	86.9	243	4.8	13.6	0.1		0.1	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.1	1.4	155	194	4393	0.09	0.09	1.24	21
Butter	20	16.0	3			80.5	16.1			2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		3	0.01	0.04		
Total	3312		2424		131.7		84.5		758		45.8		3799	11053	2.49	5.54	19.57	266

DIET I. Low PROTEIN GROUPS, September 10-October 4, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Gm.		Serv- ing	Vitamin Content per Serving				
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.		Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
CALORIC GROUP Z																			
Basic I	1400	78.7	1102	2.3	32.2	2.2	30.8	249	1.3	18.2	91	1273	2818	1.19	2.65	4.28		129	
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	901	0.52	0.40	9.90		23	
Salad B	300	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	4393	0.16	0.10	1.48		21	
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	600	0.09	0.09	1.24			
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3			0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		0.01	0.04				
Total	2207		1574		82.3		62.5	540		31.4		2724	8715	1.96	3.25	16.96		173	
CALORIC GROUP L																			
Basic I	1765	78.7	1389	2.3	40.6	2.2	38.8	314	1.3	22.9	91	1606	3552	1.50	3.34	5.39		163	
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	901	0.52	0.40	9.90		23	
Salad B	300	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	4393	0.16	0.10	1.48		21	
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	600	0.09	0.09	1.24			
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3			0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		0.01	0.04				
Total	2572		1861		90.7		70.5	605		36.1		3057	9449	2.27	3.94	18.07		207	
CALORIC GROUP G																			
Basic I	2130	78.7	1676	2.3	49.0	2.2	46.9	379	1.3	27.7	91	1940	4289	1.81	4.04	6.51		196	
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	901	0.52	0.40	9.90		23	
Salad B	300	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	4393	0.16	0.10	1.48		21	
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	600	0.09	0.09	1.24			
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3			0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		0.01	0.04				
Total	2937		2148		99.1		78.6	670		40.9		3391	10186	2.58	4.64	19.19		240	
CALORIC GROUP T																			
Basic I	2525	78.7	1987	2.3	58.1	2.2	55.6	449	1.3	32.8	91	2299	5080	2.14	4.78	7.72		232	
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	901	0.52	0.40	9.90		23	
Salad B	300	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	4393	0.16	0.10	1.48		21	
Sw. pot.	125	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	600	0.09	0.09	1.24			
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3			0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94		0.01	0.04				
Total	3332		2459		108.2		87.3	740		46.0		3750	10977	2.91	5.38	20.40		276	

TABLE 531. DIET I. HIGH PROTEIN GROUPS, October 5-October 20, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing		Vitamin Content per Serving				
	%	Gm.	%	Gm.	%	Gm.	Gm.	Gm.	%	Gm.	Gm.	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																	
Basic I	78.7	1102	2.3	32.2	2.2	30.8	249	1.3	18.2	91	1273	2818	1.19	2.65	4.28	129	
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931		0.14	0.52	8.91		
Salad A	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. ess.	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26		
Butter	16.0	3		80.5	16.1			2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04			
Total		1578		127.8		64.1	606		34.4		3006	9191	1.69	3.56	16.39	163	
CALORIC GROUP L																	
Basic I	78.7	1389	2.3	40.6	2.2	38.8	314	1.3	22.9	91	1606	3552	1.50	3.34	5.39	163	
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931		0.14	0.52	8.91		
Salad A	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. ess.	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26		
Butter	16.0	3		80.5	16.1			2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04			
Total		1874		136.2		72.1	671		39.1		3339	9952	2.00	4.25	17.50	197	
CALORIC GROUP G																	
Basic I	78.7	1676	2.3	49.0	2.2	46.9	379	1.3	27.7	91	1940	4289	1.81	4.04	6.51	196	
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931		0.14	0.52	8.91		
Salad A	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. ess.	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26		
Butter	16.0	3		80.5	16.1			2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04			
Total		2161		144.6		80.2	736		43.9		3673	10662	2.31	4.95	18.62	230	
CALORIC GROUP T																	
Basic I	78.7	1987	2.3	58.1	2.2	55.6	449	1.3	32.8	91	2299	5080	2.14	4.78	7.72	232	
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931		0.14	0.52	8.91		
Salad A	86.9	243	4.8	13.6	0.1	0.2	33	1.2	3.4	48	135	977	0.12	0.14	1.64	13	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. ess.	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26		
Butter	16.0	3		80.5	16.1			2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04			
Total		2472		153.7		88.9	806		49.0		4032	11453	2.64	5.69	19.83	266	

TABLE 532. DIET I. LOW PROTEIN GROUPS, October 5-October 20, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash	Calories per		Vitamin Content per Serving					
	%	Gm.	%	Gm.	%	Gm.	Gm.	Gm.		100 Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																	
Basic I	78.7	1102	2.3	32.2	2.2	30.8	249	1.3	18.2	91	1273	2818	1.19	2.65	4.28	129	
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90		
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.50	23	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. van.	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.62	2	
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total		1617		90.4		77.1	579		34.2		3012	9359	2.01	3.43	17.58	175	
CALORIC GROUP L																	
Basic I	78.7	1389	2.3	40.6	2.2	38.8	314	1.3	22.9	91	1606	3552	1.50	3.34	5.39	163	
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90		
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.50	23	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. van.	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.62	2	
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total		1904		98.8		85.1	644		38.9		3345	10093	2.32	4.12	18.69	209	
CALORIC GROUP G																	
Basic I	78.7	1676	2.3	49.0	2.2	46.9	379	1.3	27.7	91	1940	4289	1.81	4.04	6.51	196	
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90		
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.50	23	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. van.	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.62	2	
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total		2191		107.2		93.2	709		43.7		3679	10830	2.63	4.82	19.81	242	
CALORIC GROUP T																	
Basic I	78.7	1987	2.3	58.1	2.2	55.6	449	1.3	32.8	91	2299	5080	2.14	4.78	7.72	232	
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90		
Salad B	88.2	265	3.4	10.2		0.1	31	1.2	3.6	42	126	901	0.16	0.10	1.50	23	
Sw. pot.	63.6	80	1.1	1.4	2.8	3.5	41	1.1	1.4	155	194	4393	0.09	0.09	1.24	21	
Muf. van.	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.62	2	
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02		
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total		2502		116.3		101.9	779		48.8		4038	11621	2.96	5.56	21.04	278	

TABLE 533

DIET II. HIGH PROTEIN GROUPS, July 30-September 9, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing Gm.		Vitamin Content per Serving				
	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
CALORIC GROUP Z																	
Basic II	75.1	921	2.8	34.3	3.1	38.0	251	1.3	15.9	110	1346	2253	0.47	0.93	3.61	58	
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	1.66	2	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total		1134		76.1		44.5	399		28.2		1997	2282	0.95	1.43	10.13	60	
CALORIC GROUP L																	
Basic II	75.1	1164	2.8	43.4	3.1	48.0	318	1.3	20.2	110	1700	2850	0.59	1.18	4.56	73	
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	1.66	2	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total		1377		85.2		54.5	466		32.5		2351	2879	1.07	1.68	11.08	75	
CALORIC GROUP G																	
Basic II	75.1	1399	2.8	52.1	3.1	57.8	382	1.3	24.2	110	2045	3446	0.72	1.42	5.52	89	
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	1.66	2	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total		1612		93.9		64.3	530		36.5		2699	3475	1.20	1.92	12.04	91	
CALORIC GROUP T																	
Basic II	75.1	1673	2.8	62.5	3.1	69.1	457	1.3	29.0	110	2244	4041	0.84	1.67	6.50	104	
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	1.66	2	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Total		1886		104.3		75.6	605		41.3		2895	4070	1.32	2.17	13.02	106	

TABLE 534

DIET II. Low PROTEIN GROUPS, July 30-September 9, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Gm.		Vitamin Content per Serving				
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																		
Basic II	1226	75.1	921	2.8	34.3	3.1	38.0	251	1.3	15.9	110	1346	2253	0.47	0.93	3.61	58	
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	30	1.8	4.0	66	144	66	0.24	0.15	0.71	13	
Wh. wh.	180	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40		
Total	1626		1167		64.9		46.9	388		24.0		1977	2319	0.99	1.30	9.72	71	
CALORIC GROUP L																		
Basic II	1550	75.1	1164	2.8	43.4	3.1	48.0	318	1.3	20.2	110	1700	2850	0.59	1.18	4.56	73	
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	30	1.8	4.0	66	144	66	0.24	0.15	0.71	13	
Wh. wh.	180	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40		
Total	1950		1410		74.0		56.9	455		28.3		2331	2916	1.11	1.55	10.67	86	
CALORIC GROUP G																		
Basic II	1863	75.1	1399	2.8	52.1	3.1	57.8	382	1.3	24.2	110	2045	3446	0.72	1.42	5.52	89	
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	30	1.8	4.0	66	144	66	0.24	0.15	0.71	13	
Wh. wh.	180	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40		
Total	2263		1645		82.7		66.7	519		32.3		2676	3512	1.24	1.79	11.63	102	
CALORIC GROUP T																		
Basic II	2228	75.1	1673	2.8	62.5	3.1	69.1	457	1.3	29.0	110	2244	4041	0.84	1.67	6.50	104	
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	30	1.8	4.0	66	144	66	0.24	0.15	0.71	13	
Wh. wh.	180	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40		
Total	2628		1919		93.1		78.0	594		37.1		2875	4107	1.36	2.04	12.61	117	

TABLE 535

DIET II. HIGH PROTEIN GROUPS, September 10-October 4, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing Gm.		Vitamin Content per Serving				
	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	100 Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
CALORIC GROUP Z																	
Basic II	75.1	921	2.8	34.3	3.1	38.0	251	1.3	15.9	110	1346	2253	0.47	0.93	0.93	3.61	58
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	0.22	1.66	2
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	80	0.14	0.52	0.52	8.91	2
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	600	0.01	0.07	0.07	0.51	2
Butter	16.0	3	0.5	0.2	80.5	16.1	23	2.5	0.5	729	146	3	0.01	0.01	0.01	0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.01	0.01	0.04	
Total		1257		107.3		67.2	542		32.7		2775	2965	0.84	1.75	1.75	14.75	62
CALORIC GROUP L																	
Basic II	75.1	1164	2.8	43.4	3.1	48.0	318	1.3	20.2	110	1700	2850	0.59	1.18	1.18	4.56	73
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	0.22	1.66	2
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	80	0.14	0.52	0.52	8.91	2
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	600	0.01	0.07	0.07	0.51	2
Butter	16.0	3	0.5	0.2	80.5	16.1	23	2.5	0.5	729	146	3	0.01	0.01	0.01	0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.01	0.01	0.04	
Total		1500		116.4		77.2	609		37.0		3129	3562	0.96	2.00	2.00	15.70	77
CALORIC GROUP G																	
Basic II	75.1	1399	2.8	52.1	3.1	57.8	382	1.3	24.2	110	2045	3446	0.72	1.42	1.42	5.52	89
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	0.22	1.66	2
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	80	0.14	0.52	0.52	8.91	2
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	600	0.01	0.07	0.07	0.51	2
Butter	16.0	3	0.5	0.2	80.5	16.1	23	2.5	0.5	729	146	3	0.01	0.01	0.01	0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.01	0.01	0.04	
Total		1735		125.1		87.0	673		41.0		3474	4158	1.09	2.24	2.24	16.66	93
CALORIC GROUP T																	
Basic II	75.1	1673	2.8	62.5	3.1	69.1	457	1.3	29.0	110	2244	4041	0.84	1.67	1.67	6.50	104
Soup A	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	93	186	29	0.22	0.22	0.22	1.66	2
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	80	0.14	0.52	0.52	8.91	2
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	600	0.01	0.07	0.07	0.51	2
Butter	16.0	3	0.5	0.2	80.5	16.1	23	2.5	0.5	729	146	3	0.01	0.01	0.01	0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.01	0.01	0.04	
Total		2009		135.5		98.3	748		45.8		3673	4753	1.21	2.49	2.49	17.64	108

DIET II. Low PROTEIN GROUPS, September 10-October 4, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash	Calories per		Vitamin Content per Serving				
	%	Gm.	%	Gm.	%	Gm.	%	Gm.		100 Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
	CALORIC GROUP Z															
Basic II	75.1	921	2.8	34.3	3.1	38.0	251	1.3	15.9	110	1346	2253	0.47	0.93	3.61	58
Soup B	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.71	13
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.51	2
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total		1302		83.7		69.9	516		30.9		2693	3002	1.24	1.56	14.79	73
CALORIC GROUP L																
Basic II	75.1	1164	2.8	43.4	3.1	48.0	318	1.3	20.2	110	1700	2850	0.59	1.18	4.56	73
Soup B	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.71	13
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.51	2
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total		1545		92.8		79.9	583		35.2		3047	3599	1.36	1.81	15.74	88
CALORIC GROUP G																
Basic II	75.1	1399	2.8	52.1	3.1	57.8	382	1.3	24.2	110	2045	3446	0.72	1.42	5.52	89
Soup B	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.71	13
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.51	2
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total		1780		101.5		89.7	647		39.2		3392	4195	1.49	2.05	16.70	104
CALORIC GROUP T																
Basic II	75.1	1673	2.8	62.5	3.1	69.1	457	1.3	29.0	110	2244	4041	0.84	1.67	6.50	104
Soup B	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.71	13
Wh. wh.	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Har. beets	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.51	2
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total		2054		111.9		101.0	722		44.0		3591	4790	1.61	2.30	17.68	119

TABLE 537. DIET II. HIGH PROTEIN GROUPS, October 5–October 20, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Gm.		Serv- ing		Vitamin Content per Serving			
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	100 Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																			
Basic II	1226	75.1	921	2.8	34.3	3.1	38.0	251	1.3	15.9	0.47	0.93	110	1346	2253	0.47	0.93	3.61	58
Soup A	200	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	0.22	0.22	93	186	29	0.22	0.22	1.66	2
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	0.14	0.52	282	931	80	0.14	0.52	8.91	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	0.01	0.07	80	72	400	0.01	0.07	0.51	2
Muf. ess.	104	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	0.15	0.15	224	233	600	0.15	0.15	0.26	
Butter	20	16.0	3		80.5	16.1			2.5	0.5		729	146				0.02		
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1		312	94	3		0.01	0.04		
Total	2002		1305		129.3		71.6	590		35.9			3008	3365	0.99	1.90	15.01		62
CALORIC GROUP L																			
Basic II	1550	75.1	1164	2.8	43.4	3.1	48.0	318	1.3	20.2	0.59	1.15	110	1700	2850	0.59	1.15	4.56	73
Soup A	200	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	0.22	0.22	93	186	29	0.22	0.22	1.66	2
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	0.14	0.52	282	931	80	0.14	0.52	8.91	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	0.01	0.07	80	72	400	0.01	0.07	0.51	2
Muf. ess.	104	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	0.15	0.15	224	233	600	0.15	0.15	0.26	
Butter	20	16.0	3		80.5	16.1			2.5	0.5		729	146				0.02		
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1		312	94	3		0.01	0.04		
Total	2326		1548		138.4		81.6	657		40.2			3362	3962	1.11	2.15	15.96		77
CALORIC GROUP G																			
Basic II	1863	75.1	1399	2.8	52.1	3.1	57.8	382	1.3	24.2	0.72	1.42	110	2045	3446	0.72	1.42	5.52	89
Soup A	200	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	0.22	0.22	93	186	29	0.22	0.22	1.66	2
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	0.14	0.52	282	931	80	0.14	0.52	8.91	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	0.01	0.07	80	72	400	0.01	0.07	0.51	2
Muf. ess.	104	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	0.15	0.15	224	233	600	0.15	0.15	0.26	
Butter	20	16.0	3		80.5	16.1			2.5	0.5		729	146				0.02		
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1		312	94	3		0.01	0.04		
Total	2639		1783		147.1		91.4	721		44.2			3707	4558	1.24	2.39	16.92		93
CALORIC GROUP T																			
Basic II	2228	75.1	1673	2.8	62.5	3.1	69.1	457	1.3	29.0	0.84	1.67	110	2244	4041	0.84	1.67	6.50	104
Soup A	200	75.6	151	6.6	13.2	1.2	2.4	41	2.9	5.8	0.22	0.22	93	186	29	0.22	0.22	1.66	2
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	0.14	0.52	282	931	80	0.14	0.52	8.91	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	0.01	0.07	80	72	400	0.01	0.07	0.51	2
Muf. ess.	104	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	0.15	0.15	224	233	600	0.15	0.15	0.26	
Butter	20	16.0	3		80.5	16.1			2.5	0.5		729	146				0.02		
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1		312	94	3		0.01	0.04		
Total	3004		2057		157.5		102.7	796		49.0			3906	5153	1.36	2.64	17.90		108

TABLE 538. DIET II. Low PROTEIN GROUPS, October 5-October 20, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per		Vitamin Content per Serving					
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	100 Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																			
Basic II	1226	75.1	921	2.8	34.3	3.1	38.0	251	1.3	15.9	110	1346	2253	0.47	0.93	0.47	0.93	3.61	58
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.24	0.15	0.71	13
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	80	0.52	0.40	0.52	0.40	9.90	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.01	0.07	0.51	2
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.05	0.18	0.62	2
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600					0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.01	0.04		
Total	2040		1345		91.8		84.5	555		33.7		2981	3646	1.29	1.74	1.29	1.74	15.41	75
CALORIC GROUP L																			
Basic II	1550	75.1	1164	2.8	43.4	3.1	48.0	318	1.3	20.2	110	1700	2850	0.59	1.18	0.59	1.18	4.56	73
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.24	0.15	0.71	13
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	80	0.52	0.40	0.52	0.40	9.90	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.01	0.07	0.51	2
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.05	0.18	0.62	2
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600					0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.01	0.04		
Total	2326		1588		100.9		94.5	622		38.0		3335	4243	1.41	1.99	1.41	1.99	16.36	90
CALORIC GROUP G																			
Basic II	1863	75.1	1399	2.8	52.1	3.1	57.8	382	1.3	24.2	110	2045	3446	0.72	1.42	0.72	1.42	5.52	89
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.24	0.15	0.71	13
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	80	0.52	0.40	0.52	0.40	9.90	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.01	0.07	0.51	2
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.05	0.18	0.62	2
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600					0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.01	0.04		
Total	2677		1823		109.6		104.3	686		42.0		3680	4839	1.54	2.23	1.54	2.23	17.32	106
CALORIC GROUP T																			
Basic II	2228	75.1	1673	2.8	62.5	3.1	69.1	457	1.3	29.0	110	2244	4041	0.84	1.67	0.84	1.67	6.50	104
Soup B	220	83.4	183	4.4	9.7	1.1	2.4	31	1.8	4.0	66	144	66	0.24	0.15	0.24	0.15	0.71	13
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	80	0.52	0.40	0.52	0.40	9.90	2
Har. beets	90	78.8	71	1.3	1.2	1.6	1.4	15	3.1	2.8	80	72	80	0.01	0.07	0.01	0.07	0.51	2
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.05	0.18	0.62	2
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600					0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.01	0.04		
Total	3042		2097		120.0		115.6	761		46.8		3879	5434	1.66	2.48	1.66	2.48	18.30	121

TABLE 539

DIET III. HIGH PROTEIN GROUPS, July 30-September 9, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing Gm.		Vitamin Content per Serving				
	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
CALORIC GROUP Z																	
Basic III	64.8	582	2.8	25.1	3.3	29.6	270	1.8	16.2	150	1346	2120	0.39	0.86	5.42	51	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Loaf A	67.7	112	8.0	13.3	1.4	2.3	46	2.9	4.8	125	208	540	0.12	0.15	1.38	1	
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17		
Total		797		68.8		40.9	428		28.4		2081	2990	0.78	1.44	11.83	52	
CALORIC GROUP L																	
Basic III	64.8	741	2.8	32.0	3.3	37.7	344	1.8	20.6	150	1715	2699	0.50	1.09	6.90	65	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Loaf A	67.7	112	8.0	13.3	1.4	2.3	46	2.9	4.8	125	208	540	0.12	0.15	1.38	1	
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17		
Total		956		75.7		49.0	502		32.8		2450	3569	0.89	1.67	13.31	66	
CALORIC GROUP G																	
Basic III	64.8	920	2.8	39.8	3.3	46.9	429	1.8	25.6	150	2138	3353	0.62	1.36	8.58	81	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Loaf A	67.7	112	8.0	13.3	1.4	2.3	46	2.9	4.8	125	208	540	0.12	0.15	1.38	1	
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17		
Total		1135		83.5		58.2	587		37.8		2873	4203	1.01	1.94	14.99	82	
CALORIC GROUP T																	
Basic III	64.8	1111	2.8	48.0	3.3	56.6	517	1.8	30.8	150	2577	4045	0.74	1.64	10.35	97	
Soy bread	34.4	62	15.9	28.6	2.3	4.1	107	3.6	6.5	258	465		0.26	0.28	4.86		
Loaf A	67.7	112	8.0	13.3	1.4	2.3	46	2.9	4.8	125	208	540	0.12	0.15	1.38	1	
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17		
Total		1326		91.7		67.9	675		43.0		3312	4915	1.13	2.22	16.76	98	

TABLE 540

DIET III. Low PROTEIN GROUPS, July 30-September 9, 1945 (for legend see Table 527)

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing		Vitamin Content per Serving					
	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	100 Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																		
Basic III	64.8	582	2.8	25.1	3.3	29.6	270	1.8	16.2	150	1346	2120	0.39	0.86	5.42	51		
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40			
Loaf B	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	535	0.08	0.12	0.85	10		
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17			
Total		808		54.8		42.2	421		23.7		2067	2985	0.76	1.35	11.85	61		
CALORIC GROUP L																		
Basic III	64.8	741	2.8	32.0	3.3	37.7	344	1.8	20.6	150	1715	2699	0.50	1.09	6.90	65		
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40			
Loaf B	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	535	0.08	0.12	0.85	10		
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17			
Total		967		61.7		50.3	495		28.1		2436	3564	0.87	1.58	13.32	75		
CALORIC GROUP G																		
Basic III	64.8	920	2.8	39.8	3.3	46.9	429	1.8	25.6	150	2138	3353	0.62	1.36	8.58	81		
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40			
Loaf B	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	535	0.08	0.12	0.85	10		
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17			
Total		1146		69.5		59.5	580		33.1		2859	4218	0.99	1.85	15.00	91		
CALORIC GROUP T																		
Basic III	64.8	1111	2.8	48.0	3.3	56.6	517	1.8	30.8	150	2577	4045	0.74	1.64	10.35	97		
Wh. wh.	34.8	63	11.6	20.9	3.6	6.5	107	2.3	4.1	270	487		0.28	0.22	5.40			
Loaf B	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	535	0.08	0.12	0.85	10		
Sauce	80.2	41	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17			
Total		1337		77.7		69.2	668		38.3		3298	4910	1.11	2.13	16.77	107		

TABLE 541. DIET III. HIGH PROTEIN GROUPS, September 10–October 4, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per		Vitamin Content per Serving					
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	100 Gm.	Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																			
Basic III	898	64.8	582	2.8	25.1	3.3	29.6	270	1.8	16.2	150	1346	2120	0.39	0.86	5.42		51	
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91		1	
Loaf A	166	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38		1	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	3100	0.01	0.15	0.17		10	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	600	0.06	0.07	1.47		10	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3		0.01	0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04			
Total	1608		939	99.8		73.3	565		31.1			2922	6693	0.72	1.76	17.41		62	
CALORIC GROUP L																			
Basic III	1143	64.8	741	2.8	32.0	3.3	37.7	344	1.8	20.6	150	1715	2699	0.50	1.09	6.90		65	
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91		1	
Loaf A	166	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38		1	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	3100	0.01	0.15	0.17		10	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	600	0.06	0.07	1.47		10	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3		0.01	0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04			
Total	1853		1098	106.7		81.4	639		35.5			3291	7272	0.83	1.99	18.89		76	
CALORIC GROUP G																			
Basic III	1421	64.8	920	2.8	39.8	3.3	46.9	429	1.8	25.6	150	2138	3353	0.62	1.36	8.58		81	
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91		1	
Loaf A	166	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38		1	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	3100	0.01	0.15	0.17		10	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	600	0.06	0.07	1.47		10	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3		0.01	0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04			
Total	2131		1277	114.5		90.6	724		40.5			3714	7926	0.95	2.26	20.57		92	
CALORIC GROUP T																			
Basic III	1715	64.8	1111	2.8	48.0	3.3	56.6	517	1.8	30.8	150	2577	4045	0.74	1.64	10.35		97	
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91		1	
Loaf A	166	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38		1	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	3100	0.01	0.15	0.17		10	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	600	0.06	0.07	1.47		10	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3		0.01	0.02			
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04			
Total	2425		1468	122.7		100.3	812		45.7			4153	8618	1.07	2.54	22.34		108	

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash Gm.	Calories per 100 Gm.		Vitamin Content per Serving				
		%	Gm.	%	Gm.	%	Gm.	Gm.	Gm.		Serv- ing	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP Z																	
Basic III	898	64.8	582	2.8	25.1	3.3	29.6	270	1.8	16.2	150	1346	2120	0.39	0.86	5.42	51
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Loaf B	166	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	535	0.08	0.12	0.85	10
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	10
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total	1608		962		73.4		74.9	542		28.8		2846	6688	1.06	1.61	17.87	71
CALORIC GROUP L																	
Basic III	1143	64.8	741	2.8	32.0	3.3	37.7	344	1.8	20.6	150	1715	2699	0.50	1.09	6.90	65
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Loaf B	166	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	535	0.08	0.12	0.85	10
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	10
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total	1853		1121		80.3		83.0	616		33.2		3215	7267	1.17	1.84	19.35	85
CALORIC GROUP G																	
Basic III	1421	64.8	920	2.8	39.8	3.3	46.9	429	1.8	25.6	150	2138	3353	0.62	1.36	8.58	81
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Loaf B	166	73.4	122	4.2	7.0	0.7	1.1	40	1.5	2.9	104	172	535	0.08	0.12	0.85	10
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	10
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total	2131		1300		88.1		92.2	701		38.2		3638	7921	1.29	2.11	21.03	101
CALORIC GROUP T																	
Basic III	1715	64.8	1111	2.8	48.0	3.3	56.6	517	1.8	30.8	150	2577	4045	0.74	1.64	10.35	97
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891		0.52	0.40	9.90	
Loaf B	166	73.4	122	4.2	7.0	0.7	1.1	40	1.5	2.9	104	172	535	0.08	0.12	0.85	10
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	10
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3	0.01	0.04		
Total	2425		1491		96.3		101.9	789		43.4		4077	8613	1.41	2.39	22.80	117

TABLE 543

DIET III. HIGH PROTEIN GROUPS, October 5-October 20, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash	Calories per 100 Gm.		Vitamin Content per Serving				
		g	Gm.	%	Gm.	%	Gm.	%	Serv- ing		Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C		
CALORIC GROUP Z																	
Basic III	898	64.8	582	2.8	25.1	3.3	29.6	270	1.8	16.2	150	1346	2120	0.39	0.86	5.42	51
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91	1
Loaf A	166	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	10
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	
Muf. ess.	104	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04	
Total	1712		987		121.8		77.7	613		34.3		3155	7093	0.87	1.91	17.67	62
CALORIC GROUP L																	
Basic III	1143	64.8	741	2.8	32.0	3.3	37.7	344	1.8	20.6	150	1715	2699	0.50	1.09	6.90	65
Cas. bread	330	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91	1
Loaf A	166	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	10
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	
Muf. ess.	104	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04	
Total	1957		1146		128.7		85.8	687		38.7		3524	7672	0.98	2.14	19.15	76

TABLE 543 continued

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- Gm. ing		Vitamin Content per Serving				
	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
CALORIC GROUP G																	
Basic III	64.8	920	2.8	39.8	3.3	46.9	429	1.8	25.6	150	2138	3353	0.62	1.36	8.58		81
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91		1
Loaf A	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38		
Sauce	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17		
Car. salad	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47		10
Muf. ess.	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26		
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02		
Jam	32	27.5	9	0.5	0.2	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total		1325		136.5		95.0	772		43.7		3947	8326	1.10	2.41	20.83		92
CALORIC GROUP T																	
Basic III	64.8	1111	2.8	48.0	3.3	56.6	517	1.8	30.8	150	2577	4045	0.74	1.64	10.35		97
Cas. bread	30.8	102	17.7	58.4	2.8	9.2	212	2.3	7.6	282	931	540	0.14	0.52	8.91		1
Loaf A	67.7	112	8.0	13.3	1.4	2.3	47	2.9	4.8	125	208	330	0.12	0.15	1.38		
Sauce	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17		
Car. salad	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47		10
Muf. ess.	46.2	48	21.2	22.0	4.3	4.4	48	3.1	3.2	224	233	400	0.15	0.15	0.26		
Butter	16.0	3			80.5	16.1		2.5	0.5	729	146	600			0.02		
Jam	32	27.5	9	0.5	0.2	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total		1516		144.7		104.7	860		48.9		4386	9018	1.22	2.69	22.60		108

TABLE 544

DIET III. Low PROTEIN GROUPS, October 5-October 20, 1945 (for legend see Table 527)

	Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing Gm.		Vitamin Content per Serving				
		%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C
		CALORIC GROUP Z																
Basic III	898	64.8	582	2.8	25.1	3.3	29.6	270	1.8	16.2	150	1346	2120	0.39	0.86	5.42	51	
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	535	0.52	0.40	9.90	10	
Loaf B	166	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	62	0.08	0.12	0.85	10	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	10	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	10	
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.62	2	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600		0.01	0.02		
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total	1730		1005		81.5		89.5	581		31.6		3134	7332	1.11	1.79	18.49	73	
CALORIC GROUP L																		
Basic III	1143	64.8	741	2.8	32.0	3.3	37.7	344	1.8	20.6	150	1715	2699	0.50	1.09	6.90	65	
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	535	0.52	0.40	9.90	10	
Loaf B	166	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	62	0.08	0.12	0.85	10	
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	330	0.01	0.15	0.17	10	
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	3100	0.06	0.07	1.47	10	
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	644	0.05	0.18	0.62	2	
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	600		0.01	0.02		
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	3		0.01	0.04		
Total	1975		1164		88.4		97.6	655		36.0		3503	7911	1.22	2.02	19.97	87	

TABLE 544 continued

Total Gm.	H ₂ O		Protein		Fat		CHO and Protein		Ash		Calories per 100 Serv- ing		Vitamin Content per Serving				
	%	Gm.	%	Gm.	%	Gm.	%	Gm.	%	Gm.	Gm.	Vit. A	Thia- mine	Fla- vin	Nia- cin	Vit. C	
CALORIC GROUP G																	
Basic III	1421	64.8	920	2.8	39.8	3.3	46.9	429	1.8	25.6	150	2138	3353	0.62	1.36	8.58	81
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	535	0.52	0.40	9.90	10
Loaf B	166	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	330	0.08	0.12	0.85	10
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	3100	0.01	0.15	0.17	10
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	644	0.06	0.07	1.47	2
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	600	0.05	0.18	0.62	2
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3		0.01	0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	8565	1.34	2.29	21.65	103
Total	2253		1343		96.2		106.8	740		41.0		3926					
CALORIC GROUP T																	
Basic III	1715	64.8	1111	2.8	48.0	3.3	56.6	517	1.8	30.8	150	2577	4045	0.74	1.64	10.35	97
Wh. wh.	330	34.8	115	11.6	38.3	3.6	11.9	196	2.3	7.6	270	891	535	0.52	0.40	9.90	10
Loaf B	166	73.4	122	4.2	7.0	0.7	1.2	40	1.5	2.5	104	172	330	0.08	0.12	0.85	10
Sauce	52	80.2	42	3.4	1.8	9.5	4.9	4	1.7	0.9	119	62	3100	0.01	0.15	0.17	10
Car. salad	110	81.0	89	0.9	1.0	10.1	11.1	9	0.9	1.0	123	135	644	0.06	0.07	1.47	2
Muf. van.	122	35.0	43	6.6	8.1	12.0	14.6	39	2.3	2.8	236	288	600	0.05	0.18	0.62	2
Butter	20	16.0	3			80.5	16.1		2.5	0.5	729	146	3		0.01	0.02	
Jam	32	27.5	9	0.5	0.2	0.3	0.1	23	0.3	0.1	312	94	4365	1.46	2.57	23.42	119
Total	2547		1534		104.4		116.5	828		46.2		4365	9257				

TABLE 545

SUMMARY OF THE CALORIC CONTENT OF THE BASAL DIETS IN THE REHABILITATION PERIOD. All values are based on those given in Tables 527-44.

Caloric Group	R6		R10		R12	
	Y	U	Y	U	Y	U
Z	1984	1977	2823	2754	3056	3042
L	2336	2329	3175	3106	3408	3394
G	2704	2696	3543	3474	3776	3762
T	3035	3028	3875	3806	4108	4094
Average	2515	2508	3354	3285	3587	3573

TABLE 546

SUMMARY OF THE CARBOHYDRATE CONTENT OF THE BASIC DIETS IN THE REHABILITATION PERIOD. All values are based on those given in Tables 527-44.

Caloric Group	R6		R10		R12	
	Y	U	Y	U	Y	U
Z	332.2	337.7	450.7	452.9	476.7	483.8
L	392.8	398.2	511.2	486.4	537.2	544.3
G	455.8	461.2	574.3	576.4	600.3	607.3
T	524.2	529.7	642.7	644.9	668.7	675.8
Average	426.2	431.7	544.8	540.2	570.8	577.8

TABLE 547

SUMMARY OF THE PROTEIN CONTENT OF THE BASIC DIETS IN THE REHABILITATION PERIOD. All values are based on those given in Tables 527-44.

Caloric Group	R6		R10		R12	
	Y	U	Y	U	Y	U
Z	73.1	61.0	104.3	79.8	126.3	87.9
L	81.2	69.1	112.4	87.9	134.4	96.0
G	89.5	77.4	120.7	96.2	142.7	104.3
T	98.8	86.7	130.0	105.5	152.0	113.6
Average	85.6	73.6	116.8	92.4	138.8	100.4

TABLE 548

SUMMARY OF THE FAT CONTENT OF THE BASIC DIETS IN THE REHABILITATION PERIOD. All values are based on those given in Tables 527-44.

Caloric Group	R6		R10		R12	
	Y	U	Y	U	Y	U
Z	40.2	42.2	66.7	69.1	71.1	83.7
L	48.9	50.9	75.4	77.8	79.8	92.4
G	57.9	59.9	84.5	86.8	88.9	101.4
T	67.8	69.8	94.4	96.7	98.8	111.3
Average	53.7	55.7	80.2	82.6	84.6	97.2

TABLE 549

SUMMARY OF THE VITAMIN CONTENT OF THE BASIC DIETS IN THE REHABILITATION PERIOD. Half of the men in each group listed in the table received, in addition to the vitamins in the diet, one Hexavitamin pill per day (for composition see Table 550); the others received placebos.

	R6					R10				
	Vit. A	Thia- mine	Ribo- flavin	Nia- cin	Vit. C	Vit. A	Thia- mine	Ribo- flavin	Nia- cin	Vit. C
ZU (Basal)	3008	1.13	1.87	10.9	95	6135	1.42	2.14	16.6	106
ZY (Basal)	3022	1.10	1.98	10.9	85	6150	1.03	2.31	16.1	96
LU (+400)	3644	1.31	2.26	12.1	116	6772	1.60	2.53	17.7	127
LY (+400)	3659	1.28	2.37	12.1	106	6786	1.21	2.70	17.3	117
GU (+800)	4307	1.49	2.67	13.3	137	7434	1.79	2.93	19.0	148
GY (+800)	4315	1.47	2.77	13.3	127	7448	1.40	3.10	18.6	138
TU (+1200)	4999	1.68	3.09	14.7	160	8127	1.98	3.36	20.3	171
TY (+1200)	5014	1.66	3.20	14.7	150	8141	1.59	3.52	19.8	161
M U	3990	1.40	2.47	12.8	127	7117	1.70	2.74	18.4	138
M Y	4002	1.38	2.58	12.8	117	7131	1.31	2.91	18.0	128
M	3996	1.39	2.53	12.8	122	7124	1.50	2.82	18.2	133

	R12					R1-12				
	Vit. A	Thia- mine	Ribo- flavin	Nia- cin	Vit. C	Vit. A	Thia- mine	Ribo- flavin	Nia- cin	Vit. C
ZU (Basal)	6779	1.47	2.32	17.2	108	5307	1.34	2.11	14.9	103
ZY (Basal)	6550	1.18	2.46	16.4	96	5241	1.10	2.25	14.5	92
LU (+400)	7416	1.65	2.71	18.4	128	5944	1.52	2.50	16.1	124
LY (+400)	7186	1.36	2.85	17.5	117	5877	1.28	2.64	15.6	113
GU (+800)	8078	1.84	3.11	19.6	150	6606	1.71	2.90	17.3	145
GY (+800)	7849	1.55	3.25	18.8	138	6537	1.47	3.04	16.9	134
TU (+1200)	8771	2.03	3.54	20.9	173	7299	1.90	3.33	18.6	168
TY (+1200)	8541	1.74	3.67	20.1	161	7232	1.66	3.46	18.2	157
M U	7761	1.75	2.92	19.0	140	6289	1.62	2.71	16.7	135
M Y	7532	1.46	3.06	18.2	128	6222	1.38	2.85	16.3	124
M	7646	1.60	2.99	18.6	134	6255	1.50	2.78	16.5	130

TABLE 550

VITAMIN SUPPLEMENTS SUPPLIED DAILY DURING THE REHABILITATION PERIOD to each of the 16 men in the vitamin-supplemented group (H) in the form of Hexavitamin tablets, U.S.P. The other 16 men received placebos.

Vitamin A	2500 I.U.	Niacin amide	10.0 mg.
Thiamine	1.00 mg.	Ascorbic acid	37.5 mg.
Riboflavin	1.50 mg.	Vitamin D	200 I.U.

TABLE 551
 REPRESENTATIVE DIETS SERVED DURING THE LATE REHABILITATION PERIOD.

Food Served	Gm. Weight of Portion	Food Served	Gm. Weight of Portion	Food Served	Gm. Weight of Portion
October 31, 1945 (R14)					
Pear sauce	100	Milk	1440	Pork chop	90
Corn flakes	50	Country sausages ..	115	Potatoes	150
Butter	60	Sweet potatoes ...	250	Carrots	75
Sugar	20	Green beans	120	Vegetable salad ..	50
Jam	30	Salad greens	120	Chocolate sundae .	90
Bread	530	Ice cream	120	Egg salad	40
November 8, 1945 (R15)					
Fried egg	50	Butter	85	Turkey	60
Bacon	20	Meat pie	165	Dressing	70
Milk	1440	Beets	135	Potatoes, whipped.	120
Sugar	30	Potatoes	150	Salad greens	50
Jam	30	Waldorf salad	140	Cranberry sauce ..	30
Bread	530	Jello, custard sauce	230	Ice cream	90
Rice krispies	50	Vegetable soup ...	270	Peanut butter	70
November 28, 1945 (R18)					
Pineapple juice ...	100	Tomato juice	200	Pork chop	90
Jam	30	Meat pie	350	Potatoes, escaloped	125
Butter	30	Potatoes	200	Peas	100
Syrup	65	Cabbage salad	150	Rolls	70
Milk	480	Bread	120	Butter	10
Bread	180	Butter	10	Milk	480
Pancakes	200	Milk	480	Carrot salad	75
		Fruit cup	120	Sundae	90
December 18, 1945 (R20)					
Rice krispies	50	Creamed ham	130	Roast mutton	30
Pear sauce	100	Baked potato	110	Sweet potatoes ...	90
Milk	720	Spinach	60	Corn	70
Bread	200	Carrot, lettuce salad	45	Vegetable salad ..	125
Butter	25	Jam	20	Cherry pie	130
Sugar	20	Sherbet (raspberry)	75		

TABLE 552
 COMPOSITION OF REPRESENTATIVE DIETS SERVED DURING THE LATE REHABILITATION PERIOD. All values were calculated from standard tables of food composition.

	Protein			Fat			Carbohydrate			Total Cal.
	Gm. Wt.	Cal.	% Total Cal.	Gm. Wt.	Cal.	% Total Cal.	Gm. Wt.	Cal.	% Total Cal.	
October 31 .. (R14)	189.3	757.2	14.5	225.4	2028.6	38.7	612.7	2450.8	46.8	5236.6
November 8.. (R15)	175.5	702.0	13.5	235.0	2115.0	40.6	597.2	2388.8	45.9	5205.8
November 28. (R18)	159.5	638.0	13.2	176.9	1592.1	32.8	655.0	2622.4	54.0	4852.5
December 18. (R20)	73.5	294.0	11.2	83.7	753.3	28.6	396.7	1586.8	60.2	2634.1

APPENDIX III

Wartime Diets and Rations

TABLE 553

FOOD SERVED TO FRENCH PRISONERS OF WAR INTERNED IN GERMANY DURING WORLD WAR I. Based on the report of Richet and Mignard (1919).

	Mon- day	Tues- day	Wednes- day	Thurs- day	Fri- day	Satur- day	Sun- day	Daily Average
Cereal								
Wt.	485	560	435	485	510	435	560	496
Cal.	1675	1950	1500	1675	1775	1500	1950	1718
Potatoes								
Wt.	60			60				17
Cal.	50			50				14
Fat								
Wt.	50				55			15
Cal.	366				403			110
Meat								
Wt.	100	155	100	100	100	150	50	108
Cal.	180	250	180	180	180	250	180	200
Coffee								
Wt.	10	10	10	10	10	10	10	10
Cheese								
Wt.				50			50	14
Cal.				170			170	48
Fruits and vegetables								
Wt.			400		60	400		123
Cal.			108		34	108		36
Jam								
Wt.			75					11
Cal.			218					31
Sweets								
Wt.	22	22	22	22	22	22	22	22
Cal.	88	88	88	88	88	88	88	88
Total								
Wt.	727	747	1042	727	757	1017	692	816
Cal.	2359	2288	2094	2163	2480	1946	2388	2245

TABLE 554

FOOD AVAILABLE TO GERMAN CIVILIANS DURING THE LATTER PART OF
WORLD WAR I as reported by Loewy (1919).

	April 1916		July 1916		April 1917	
	Prot.	Cal.	Prot.	Cal.	Prot.	Cal.
Bread and rolls	17.7	672.6	18.3	663.8	17.9	674.0
Baked goods	1.1	41.9	0.8	31.8	0.4	17.8
Flour, pastry	5.3	168.9	6.8	216.4	7.4	233.4
Potatoes	11.2	503.8	8.2	370.0	7.3	327.9
Butter and fats		229.8		202.0		144.0
Meat and sausage	7.6	101.6	6.8	90.3	9.7	129.4
Meat preserves	2.6	17.6	2.4	16.1	0.3	1.9
Fish, smoked foods	4.2	29.1	4.6	31.9	2.4	16.4
Fish preserves	1.0	6.7				
Eggs	2.5	33.4	1.4	18.9	1.5	19.4
Milk (liters)	9.1	178.7	9.9	194.5	8.7	172.0
Canned milk	1.0	22.5	0.6	13.7		
Cheese	3.6	36.3	5.0	50.3	3.3	33.3
Vegetables, fruits	0.6	28.4	1.4	71.4	0.8	40.2
Vegetables, fruits (canned)	0.4	29.0	0.2	15.1	0.2	14.7
Marmalade		59.3		61.9		41.4
Sugar		157.9		167.7		119.6
Cocoa, sweets	0.4	25.5	0.3	16.7		
Total	68.3	2343.0	66.7	2232.5	59.9	1985.4

TABLE 555

COMPOSITION OF AVERAGE DAILY RATIONS ISSUED BY THE GERMANS TO RUSSIAN AND BRITISH PRISONERS IN A CAMP AT TOST, GERMANY, DURING WORLD WAR II. Most of the foods were cooked together in the form of a stew which was served twice a day. A cup of "coffee" (150 cc.), bread, and fat made up the daily menu. All values are only rough approximations, erring probably on the high side since they are based on the food lists supplied by the German authorities. The statement is made that the weights were "usually moderately correct, but much of the food was bad, and all was weighed unclean." From Leyton (1946).

	Average Weight before Cooking (gm.)	Protein (gm.)	Fat (gm.)	Carbo- hydrate (gm.)	Calories
Bread	330	35	2	148	742
Potatoes	400	9	0	77	344
Vegetables	400	7	0	22	116
Fat	25	0	21	0	189
Sugar	25	0	0	20	80
Jam	25	0	0	13	52
Meal	15	1	0	7	32
Meat	25	5	4	0	56
Total	1245	55	27	287	1611

TABLE 556

COMPOSITION OF FOOD SERVED IN A PARISIAN INSANE ASYLUM DURING APRIL 1941. This food was representative of that served during the early part of the German occupation. The adult inmates lost from 10 to 18 kg. From Gounelle *et al.* (1941a).

	1st Day	2d Day	3d Day	4th Day	5th Day	6th Day	7th Day	Average
CHO (gm.) . . .	233.2	230.8	313.5	238.8	253.2	259.7	265.4	256.3
Fat (gm.)	15.6	15.6	50.3	28.4	15.0	15.8	17.3	22.6
Protein (gm.) .	44.0	45.4	85.8	51.7	58.1	47.1	50.0	54.6
Total calories per day	1249	1246	2050	1418	1380	1369	1418	1447
Vitamin A (I.U.)	2898	8273	25268	1205	2950	1906	4472	6710
Thiamine	1.09	1.26	1.17	1.02	1.49	1.00	1.51	1.22
Riboflavin73	1.05	.71	.78	.79	.77	.88	.82
Vitamin C (mg.)	165	309	95	185	203	153	233	192
Vitamin D (I.U.)	2	2	3752(?)	2	2	2	3	538(?)
Ca (gm.)31	.51	.37	.33	.54	.34	.36	.40
P (mg.)	776	804	877	783	1116	772	941	867
Fe (mg.)	12	14	16	12	12	13	16	14

TABLE 557

NUTRITION IN A PARIS INSANE ASYLUM, APRIL TO DECEMBER 1941. The mean nutrient value of the daily diet received by men patients, estimated for periods of 4 to 11 days for each period indicated, is summarized below for the year 1941. The diet from August 1940 until the end of 1941 was substantially constant. Prior to July 1940 the patients were well nourished. The general level of the vitamins in the daily diet was estimated to be surprisingly good: vitamin A and carotenoids, 11,850 I.U.; thiamine, 1.7 mg.; riboflavin, 532 Sherman-Bourquin units (about 1.3 mg.); nicotinic acid, 15.9 mg.; ascorbic acid, 142 mg. This diet was associated with progressive severe emaciation, edema, coma, and a rise in mortality from one death per month to 11 deaths per month after a year and a half. Data from Bachet (1943, pp. 26-29).

	April	September	October	December	M
Calories	1436	1759	1862	1740	1699
Carbohydrate (gm.)	256	328	340	325	312
Total protein (gm.)	54	61	68	77	65
Animal protein (gm.) . .	18	16	20	11	16
Total fat (gm.)	23	23	26	24	24
Animal fat (gm.)		5	10	7	7
Calcium (gm.)	0.40	0.62	0.49	0.50	0.50
Phosphorus (gm.)	0.87	1.22	1.18	0.89	1.04
Iron (gm.)	14	17	18	23	18

TABLE 558

COMPOSITION OF FOODS DISTRIBUTED DAILY IN THE NETHERLANDS TO ADULTS (sedentary individuals; those doing harder work received extra food). From Dols and van Arcken (1946).

Year and Quarter	Calories	CHO (gm.)	Protein (gm.)	Fat (gm.)	Ca (gm.)	P (gm.)	Fe (mg.)	Vitamins		
								A (I.U.)	B ₁ (mg.)	C (mg.)
1941										
II	1828	289	59	42	0.61	1.28	15	2723	1.05	36
III	1852	310	57	38	0.62	1.30	15	2169	1.09	109
IV	1828	308	58	36	0.66	1.30	14	3802	1.11	92
1942										
I	1810	299	57	38	0.61	1.27	14	1135	1.07	50
II	1699	277	55	35	0.63	1.25	14	2691	1.03	55
III	1750	299	56	32	0.62	1.28	14	2124	1.08	113
IV	1876	326	57	32	0.67	1.35	15	4917	1.18	110
1943										
I	1822	316	52	32	0.55	1.25	14	1903	1.10	65
II	1724	317	53	23	0.56	1.25	15	2744	1.12	76
III	1783	323	52	26	0.61	1.30	15	2133	1.17	137
IV	1784	321	51	28	0.61	1.26	15	3690	1.14	101
1944										
I	1656	299	45	26	0.54	1.21	14	1633	1.12	63
II	1585	288	48	21	0.56	1.22	14	2601	1.10	70
III	1529	279	45	22	0.59	1.21	14	2261	1.07	109
Average	1752	304	53	31	0.60	1.27	14	2609	1.10	85

TABLE 559
AVERAGE CALCULATED DAILY FOOD INTAKE* DURING 1940 AND 1941 IN
THE CENTRAL PRISON AT LOUVAIN, BELGIUM, as gm. From
Simonart (1948, pp. 207-20).

	Animal Protein	Vegetable Protein	Fat	Carbo- hydrate	Total Calories
Before the war	24	92	60	600	3370
October 1940	13	36	34	344	1775
March 1941	16	39	20	354	1860
May 1941	15	34	17	327	1660
July 1941	10	43	25	303	1647

* These values do not take into account losses which occur during the preparation of food and the fact that some dietary items, especially the vegetables, are not assimilated completely. The actual daily caloric intake during the period of semi-starvation was estimated as 1400 to 1500 Cal.

TABLE 560
CALORIC CONTENT OF A WEEK'S RATION AT THE CONCENTRATION CAMP
AT DACHAU, GERMANY (from Lamy *et al.*, 1948, p. 41 after Rosenberger).

	September 1944	April 1945		September 1944	April 1945
Soup, noon	1050	1750	Jam	100	
Soup, evening	225	300	Sugar	280	
Bread	4200	1680	Cheese	25	
Potatoes	1050		Total	7120	3730
Margarine	70		Per day	1017	533
Sausage	120				

TABLE 561
ESTIMATED NUTRIENTS IN DIET SUPPLIED FOR 7 MONTHS IN 1942 AT CHANGI PRISONER-OF-WAR CAMP, SINGAPORE. The men at this camp did forced labor and most had malaria. On release in 1945 numerous cases of amblyopia, sensory spinal ataxia, nerve deafness, spastic paraplegia, and peripheral neuritis were found. Data from Burgess (1946, *Lancet* 2, p. 411, see Spillane, 1947, p. 147).

	March	April	May	June	July	August	September	M
Total calories	2060	2060	2263	2222	2315	2466	2548	2276
Non-fat calories	1874	1911	2058	2017	2120	2271	2251	2072
Protein (gm.)	41	40	45	50	47	43	48	44.9
Thiamine (mg.)	0.39	0.41	0.59	0.73	0.55	0.49	0.68	0.55
Riboflavin (mg.)	0.50	0.43	0.47	0.57	0.52	0.48	0.56	0.50
Niacin (mg.)	6.8	7.0	11.1	14.3	11.0	8.5	11.2	10.0

TABLE 562

AVERAGE DAILY DIET FOR CANADIAN SOLDIERS HELD PRISONERS OF WAR BY THE JAPANESE IN THE HONG KONG AREA. This tabulation is for foods as supplied to the cookhouse from all sources but makes no allowance for preparation, cooking, or table losses. The meat issue, for example, included bone, and the fish issue "about half the time was so rotten as to be almost entirely inedible." Food values calculated from the tables of the Manual of Hygiene of the Indian Army. Carbohydrate, protein, and fat in gm. Qualitatively, the diet seemed

to be highly deficient in at least some members of the B complex. Subsistence on the diet was associated with a multitude of neurological changes, many of severe degree, as well as edema, emaciation, and other signs of severe caloric deficit. For the total period 1942-45 the mean caloric content of the daily diet was 2305 Cal., provided by 451 gm. of CHO, 60 gm. of protein, and 29 gm. of fat. Material summarized from Crawford and Reid (1947, Table 1).

	1942			1943			1944			1945		
	CHO	Prot.	Fat	CHO	Prot.	Fat	CHO	Prot.	Fat	CHO	Prot.	Fat
January	190	21	3	527	72	43	2766	63	25	2546	40	19
February	403	52	18	547	83	37	2946	66	29	2729	39	29
March	413	58	22	583	75	39	3067	50	25	2006	47	31
April	509	74	23	575	83	32	2989	63	33	2275	47	29
May	485	72	19	542	73	37	2864	407	51	2173	49	41
June	369	61	22	442	63	29	2332	426	62	2327	48	18
July	422	65	20	452	59	23	2326	412	64	2431	58	27
August	411	51	6	478	62	22	2412	413	54	2071		
September	431	58	6	474	56	36	2548	413	58	2350		
October	511	68	33	466	67	33	2487	395	48	2069		
November	440	58	55	511	64	24	2566	414	61	2278		
December	502	80	59	554	68	28	2804	416	60	2411		
M	424	60	24	513	69	32	2616	435	58	2260	47	28
												2112

DAILY FOOD CONSUMPTION OF THE INTERNEES AT THE POOTUNG CIVIL ASSEMBLY CENTER OUTSIDE SHANGHAI, 1943-45. The calories provided in the official Japanese ration, together with that secured from International Red Cross packages, are given. The values for the official rations are based on the weights of the food delivered to the camp commissary. By means of a scale that had been secured surreptitiously, the internees checked on the weights of the food reported by the Japanese officers as having been delivered to the camp. As soon as the Japanese suspected that their deliveries were being checked, the recorded weights and those obtained by the internees agreed very closely. Interned in this camp from October 1943 through August 1945 were 980 men and 100 women. A few men raised vegetables but this constituted such a small fraction of the entire intake that it has been omitted from the calculations. Data for this table supplied by Drs. Robert Salmon and K. I. Graham.

Year and Month	Caloric Content			Caloric Components			Minerals			Vitamins			
	Total	Jap.	I.R.C.	CHO (gm.)	Fat (gm.)	Prot. (gm.)	Ca (mg.)	P (gm.)	Fe (mg.)	Vit. A (I.U.)	Thiamine (mg.)	Ribo-flavin (mg.)	Vit. C (mg.)
1943													
February	2321	2117	204	387	44	80	0.40	1.1	15				
September	2046	1842	204	377	18	86	0.34	1.4	22				
October	2009	1786	223	355	22	88	0.35	1.6	29	7549	1.3	0.70	37
November	1916	1682	233	352	18	82	0.46	1.4	33	9591	1.4	0.64	45
December	2164	1961	203	381	28	91	0.47	1.6	31	7711	1.5	0.56	54
1944													
January	2105	1950	128	387	20	88	0.44	1.4	34	4110	1.4	0.68	48
February	2420	1964	456	406	60	100	0.52	1.6	36	42802	1.6	0.68	52
March	2247	1886	361	404	25	87	0.50	1.6	35	13906	1.4	0.54	58
April	1940	1798	142	362	17	77	0.45	1.4	33	9849	1.2	0.40	43
May	1814	1758	57	342	16	71	0.44	1.3	28	12636	1.3	0.57	39
June	1754	1736	20	317	16	70	0.39	1.3	25	4488	1.2	0.45	34
July	1792	1789	3	356	15	72	0.36	1.3	23	2253	1.4	0.75	30
August	1911	1847	64	335	16	73	0.34	1.3	23	3613	1.3	0.75	32
September	1672	1672		339	14	65	0.29	1.2	21	5080	1.1	0.46	29
October	1796	1794		366	10	64	0.33	1.3	21	7303	1.1	0.51	42
November	1975	1957	19	375	18	73	0.39	1.4	25	6978	1.2	0.52	40
December	2672	1965	669	441	52	100	0.45	1.8	29	7229	1.6	0.54	34
1945													
January	2070	1775	295	386	19	82	0.35	1.6	26	13059	1.4	0.52	28
February	2029	1776	253	384	18	77	0.25	1.5	25	10862	1.3	0.59	22
March	2079	1848	226	396	17	79	0.39	1.5	31	16864	1.4	0.76	24
April	2034	1891	120	368	25	77	0.43	1.4	34	14065	1.5	0.75	29
May	2015	1890	72	380	26	82	0.52	1.1	23	3267	1.4	0.35	29
June	1892	1888	4	397	17	71	0.26	0.8	13	1593	0.8	0.32	20
July	2638	1752	886	490	49	103	0.49	1.6	31	2639	1.2	0.48	44
Average	2055	1847	220	378	24	81	0.40	1.4	27	9429	1.3	0.57	37

TABLE 564

RED CROSS PARCELS SUPPLIED TO BRITISH PRISONERS. Composition of weekly rations sent to each British prisoner during the period of his captivity at Tost, Germany, during World War II. No indication is given as to how accurate these values are or how regularly the rations were supplied. This ration was in addition to that issued by the Germans, and the two combined were sufficient to maintain the British in weight equilibrium. From Leyton (1946).

	Average Amount (gm.)	Protein (gm.)	Fat (gm.)	Carbo- hydrate (gm.)	Calories
Meat	480	96	48	0	806
Butter	240	0	184	0	1696
Cheese	90	24	30	0	390
Bacon	90	9	54	0	540
Fish	240	40	0	0	160
Sugar	240	0	0	220	880
Milk powder	400	100	100	150	1900
Biscuits	360	36	30	252	1398
Chocolate	120		30	70	490
Jam	240			100	400
Dried fruits	90	4		72	283
Oatmeal	60	10	4	40	240
Total	2650	319	480	904	9183
Per day	378	45.5	68.5	129.1	1312

TABLE 565

COMPOSITION OF CARE (COOPERATIVE FOR AMERICAN REMITTANCES TO EUROPE) PACKAGES. A maximum of three packages could be sent to any one family in any part of Austria, Belgium, Czechoslovakia, Finland, France, Greece, Italy, the Netherlands, Norway, Poland, or in the American, British, and French zones of Germany and all sectors of Berlin. The packages were made available for civilian feeding under this program in March 1946. There were five packages differing in the types of meat, vegetables, cereals, sweets, etc. They contained an average of 40,000 Cal.

	Net Weight in Lbs.
Solid meat, stew, and hashes	9.8
Cereal and biscuits	6.5
Sugar and candy	3.9
Fruit jam and pudding	3.6
Vegetables	2.3
Cocoa, coffee, and beverage powders	1.1
Evaporated milk	0.8
Preserved butter	0.5
Cheese (included in one assortment)	0.4
Soap, chewing gum, matches, etc.	1.2

Some Notable Famines in History

THE following list of famines must be considered far from complete, particularly for all parts of the world except the British Isles, Northwestern Europe, and the Mediterranean basin. In Asia, the traditional home of famines, only India has been covered by any more than a casual examination of the historical records. Purely local famines have been omitted except in a few cases when they have involved notable conditions and have been unusually well authenticated. Nor have we included those periods of food scarcity which, though attended by great suffering and elevated mortality, did not produce real famine conditions. Loveday's (1914) statement with regard to India could be applied to many other regions: "It would probably be possible to find proof of local failure of crops, and ensuing distress, in some part of India in every year of the last 2 centuries" (p. 135).

In general, famines caused directly by military action have been omitted; every notable siege and practically every major military campaign have involved starvation, either among the military forces, as in the case of the crusaders in the First Crusade and in Napoleon's army retreating from Moscow, or among the inhabitants of the unfortunate city under attack, as in the capture of Athens by Demetrius the First and in the siege of Paris in the Franco-Prussian War.

It is often difficult to separate the direct war origin from the more natural causes of famine. In 1527 the ruler of Sind, Jam Ninda, ordered the destruction of standing grain as a defensive measure; the resulting famine was almost purely the result of this measure and was relieved in about 6 months. In 1540 Mirza Shah Humayun prohibited food imports and forbade the sowing of grain in the areas adjacent to the expected line of attack; the resulting scarcity was added to the effects of 2 succeeding years of natural crop failure. Both these famines in Sind have been included in this list.

For the majority of the famines listed here the authenticating references are given by Howe (1631), Penkethman (1748), Short (1749), Gaspard (1821), Walford (1879), Danvers (1886), Loveday (1914), and Fisher (1927). The original sources, however, will be found singularly barren of the kind of detail useful to the scientist in his attempt to analyze the natural history of starvation in the individual or to appraise the behavioral and social consequences.

The concentration of the historian on political and, in the narrow sense, economic concerns seldom provides adequate information on the homely matters of the personal lives of ordinary people. Famine is generally depicted only in terms

of food prices and the administrative decisions made because of the political requirements involved. The intensity of the various famines and food shortages must be judged largely from quoted grain prices (a dubious basis in view of general fluctuations in the value of money) and from the vigor of such steps as food requisitioning and public distribution. Occasionally there are firsthand statements which lend color but not quantitative bases to the picture. Even such descriptions as that of Kashmir in the year 917-18, given in Kalhana's *Rajatarangini* (cited by Loveday, 1914, p. 11), are not common: "One could scarcely see the water in the Vitasta [Jehlam], entirely covered as the river was with corpses soaked and swollen by the water in which they had long been lying. The land became densely covered with bones in all directions until it was like one great burial ground, causing terror to all beings."

The primary cause of most famines has been simply crop failure engendered by unfavorable weather, usually drought. But the resulting suffering has been determined in large measure by prevailing methods of distribution. Loveday (1914) emphasized the dislocation of employment. Whenever decreased food production coincided with changing economic conditions, some segments of society were penalized in the competition to exchange their goods and services for food and so suffered from famine. It is of interest that until recently some writers on this subject have tended to blame the interference of government with the natural processes of free economic exchange for many of the worst effects of food shortage, if not for the food shortages themselves. Indeed this seems to have been the theme of Walford's (1879) book. Walford cited some rather convincing examples, and it would be possible to add more recent cases, the Ukraine in 1933 being a notable instance. On the other hand, once a food shortage has been established, there is little doubt that governmental intervention is becoming more effective, and in general, modern famine relief measures are attended by at least some degree of success.

It has seemed desirable to separate, in the following list, the famines of India from those of the rest of the world. Roughly a fourth of all the famines included here took place in India, and it seemed useful to indicate the different affected areas in the subcontinent.

NOTABLE FAMINES OUTSIDE INDIA

Year	Region	Year	Region
1708 B.C.	Egypt, Palestine, 7 years (Genesis 41)	175	Rome
493 B.C.	Rome	192	Ireland, emigration
436 B.C.	Rome	228	Scotland
A.D. 6	Rome	238	Scotland
10-15	Ireland	259	Wales
42	Egypt, Judea	272	Britain
51	Greece	288	Britain
54	England	298	Wales
76	Ireland	306	Scotland, 4 years
104	England, Scotland	307	Asia Minor
107	Britain	310	England, 40,000 died
119	Britain	325	Britain
151	Wales	331	Antioch, Constantine sent much relief food
160	England	336	Syria
173	England	370	Phrygia

NOTABLE FAMINES OUTSIDE INDIA *continued*

<i>Year</i>	<i>Region</i>	<i>Year</i>	<i>Region</i>
381	Antioch	883	Italy
410	Rome	887	England, 2 years
434	Italy	890	Scotland
439	Britain	895	Ireland, 3 years
446	Constantinople	896	Paris, 4 years
450	Italy, cannibalism	898	France
466	Britain	900	England
475	Northern Europe	931	Wales
480	Scotland	932	France
484	North Africa	936	Scotland, 4 years
515	Britain	945	France, 2 years
520	Venice, relief sent by Theodoric the Great	946	Italy
523	Scotland	954	Britain, 4 years
527	North Wales	962	England
531	South Wales	963	Ireland, 2 years
535	Ireland, 4 years	968	Germany, Scotland, and other regions
537	Scotland, Wales	969	England
538	Italy	975	England, Paris
547	Italy	976	England, the <i>miela hungor</i> of John of Bromton
576	Scotland	987	Albania
586	France	988	England, 2 years
590	England	1004	England, 2 years
592	England	1008	Wales
600	France, 5 years	1012	England, Germany
605	England	1016	Europe
625	Britain	1024	Russia
664	Ireland	1025	England
667	Scotland	1031	England
669	France	1035	Byzantium
669	Ireland, 2 years	1042	England, 7 years
680	Britain	1047	Ireland
683	Syria, Libya	1047	Scotland, 2 years
695	England, Ireland, 3 years, cannibalism	1050	England
703	Italy, 3 years	1051	Mexico, Toltecs migrated
712	Wales	1053	England, 2 years
718	Syria	1058	Poland
730	Britain	1064	Egypt, 7 years, cannibalism
746	Wales	1068	England
748	Scotland	1069	England, 9 years, chiefly from desolation by the Normans, cannibalism
759	Ireland, several years	1078	Constantinople
768	Ireland	1080	Denmark
772	Ireland	1086	England, 2 years
791	Wales	1087	Denmark
792	Scotland	1093	England
793	England	1099	England
803	Scotland	1100	Antioch
822	England, Scotland, 2 years	1106	England
824	Ireland, 2 years	1111	England
836	Wales	1116	Ireland, cannibalism
845	Bulgaria	1117	England
850	Paris	1120	Palestine
851	Italy, Germany	1121	England, 2 years
856	Scotland, 4 years	1123	France, Germany, 2 years
863	Scotland	1124	England, 3 years
868	Paris	1130	Rome, 2 years
872	England	1135	England, 3 years
873	Paris	1141	England, more or less for 12 years
879	Europe, Mediterranean area		

NOTABLE FAMINES OUTSIDE INDIA *continued*

<i>Year</i>	<i>Region</i>	<i>Year</i>	<i>Region</i>
1146	France	1410	Ireland
1151	Europe, Palestine, 2 years	1427	England
1153	Ireland	1429	Scotland
1154	England	1433	Ireland
1157	Italy	1437	England, 4 years
1162	Many regions	1440	Scotland
1175	England	1442	Sweden
1176	Wales	1443	Siam
1183	England, Wales	1447	Ireland
1188	Ireland	1486	England
1193	England, France, 4 years	1491	Ireland, England
1200	Ireland, Egypt	1494	England
1203	England, Ireland	1497	Ireland
1209	England	1521	England
1215	Russia	1522	Ireland
1224	England	1523	England
1227	Ireland	1527	England
1230	Rome	1528	Venice
1235	England, 20,000 dead in London	1540	Sardinia
1239	England, cannibalism	1545	England
1243	Hungary, from the Tartar invasion	1549	England
1248	Germany, England	1556	England, 3 years
1257	England, 2 years, 20,000 dead in London	1563	London
1262	Ireland	1563	Britain, Ireland
1268	Sicily, Austria	1581	Persia
1271	England, Ireland	1586	England, Ireland, Hungary, cannibalism
1281	Poland	1588	Ireland, 2 years, cannibalism
1286	England, shortage for 23 years	1591	Italy
1289	England	1594	England, Hungary
1294	England, 2 years	1595	Italy, Germany, and other regions, 2 years
1295	Ireland	1595	England
1297	Scotland	1600	Russia, 3 years, 500,000 dead of famine and pestilence
1298	England	1601	Ireland, 3 years, cannibalism
1299	Persia	1610	Saxony
1302	Britain	1630	England
1314	England, Ireland, Poland, Baltic States	1649	Scotland, northern England
1316	England, Ireland, cannibalism, edict against beermaking	1650	Ireland, 2 years
1317	Ireland, caused by Bruce's invasion	1656	Rome, 2 years
1321	England, sometimes called the last great famine in England	1690	Ireland, Italy
1332	Ireland	1693	France
1335	England	1694	Scotland, England, 5 years
1336	Scotland	1700	England
1337	China	1709	France, Scotland, England
1339	Ireland	1711	Austria
1341	England, Scotland	1727	Ireland, 3 years
1347	Italy, two thirds of the population dead of famine and pestilence	1739	France
1350	Barbary	1739	Ireland, 2 years
1353	England, France	1740	England, 2 years
1355	England	1741	Scotland
1358	England	1748	England
1361	Poland	1765	Ireland
1369	England	1766	Scotland
1374	Italy	1769	France, a stated 5 per cent of population dead
1390	England, 3 years	1770	Bohemia, a stated 168,000 dead of famine and pestilence

NOTABLE FAMINES OUTSIDE INDIA *continued*

<i>Year</i>	<i>Region</i>	<i>Year</i>	<i>Region</i>
1770	Russia, Poland	1847	France
1771	Italy	1851	Russia
1773	Russia, 2 years	1855	Russia
1775	Cape Verde Islands, 16,000 dead	1867	Russia, 2 years
1777	Russia	1871	Persia
1786	Russia	1874	Asia Minor, 2 years, 150,000 dead
1789	France	1877	Egypt, Morocco, Brazil, and Russia
1795	England	1877	Northern China, appalling conditions, 9 million dead
1801	Britain	1878	Morocco, Kashmir
1812	England, Ireland	1884	Russia
1813	Poland, Norway	1891	Russia, 2 years, \$700,000 from U.S.A. for relief
1817	Central France	1898	Russia
1822	Ireland	1906	Russia, 22 per cent of population affected
1826	Rangoon	1911	Russia, 25 million people affected
1830	Russia, several years	1916	China
1831	Ireland, £114,000 from England for relief	1919	Russia, 5 years, \$60,000,000 from U.S.A. for relief
1832	Mongolia	1929	China (Hunan Province), 2 million dead
1845	Ireland, £850,000 for relief	1933	Ukraine
1845	Russia, 2 years		
1846	Belgium		
1846	Ireland, 5 years, the "great potato famine," £10,000,000 for relief, population reduced over 2 million by deaths and migration		

FAMINES IN INDIA

<i>Year</i>	<i>Region</i>	<i>Year</i>	<i>Region</i>
503-443 B.C.	General, almost continuous, legend	1540-43	Sind, 3 years
A.D. 297	Magahda, legend	1554-55	Delhi, Agra, and Bajama districts
445	Kashmir	1556	Hindustan, cannibalism
650	Not specified	1574	Gujarat, plague
917-18	Kashmir	1576	Delhi
941	Not specified	1577	Kutch
1022	Hindustan	1592	Sholapur district, date uncertain
1033	Hindustan	1594-98	Central India, Hindustan, 3 or 4 years, very bad, also plague
1052	Hindustan, 7 years	1613-15	Punjab
1116-19	Deccan, Burhampur	1623	Gujarat, Ahmedabad
1148	General, 11 years	1628-29	Hoozoor, cannibalism
1200	Bombay, 12 years according to legend	1629-30	Deccan, almost all the dominions of Shah Jehan
1259	Bombay	1650	Ahmedabad, several years
1291	Delhi, Siwalik district	1660	Aurangzib's dominions, Punjab, one of the worst on record
1296-1317	Delhi, date uncertain	1676-77	Hyderabad
1343-45	Delhi, also plague	1685	Deccan, Hyderabad
1351-63	Kashmir, date uncertain	1702-4	Bombay, Deccan, a claimed 2 million dead
1396	Deccan, 12 years, one of the worst on record	1709	Bombay district
1412	Ganges basin, 2 years	1718	Bombay district
1423	Deccan	1737	Bombay district
1471-72	Bijapur, Bahmini Kingdom, and Bombay	1739	Bombay district
1494	Delhi	1746-47	Bombay district, Kutch
1500	Delhi	1759	Bombay district
1520	Bombay	1770	Bengal, a third of the population reported dead
1527	Sind	1781	Madras
1540	Coromandel Coast		

FAMINES IN INDIA *continued*

<i>Year</i>	<i>Region</i>	<i>Year</i>	<i>Region</i>
1782	Bombay and Madras districts, 2 1/2 years	1838	Bombay
1783	Bengal, Bellary, United Provinces, Kashmir, Rajputana	1853-55	Bellary, southern Madras, Deccan, Rajputana, part of Bombay district
1787	South Mahratta country, locusts	1860-61	Parts of Northwestern Provinces, Punjab, Rajputana, Kutch
1790-92	Bombay, Hyderabad, Gujarat, northern Madras, Kutch, Orissa	1862	Deccan
1799-1801	Northwestern Provinces, Bombay, Central India	1866-67	Orissa, Behar, Ganjam, Bellary, Hyderabad, southern Mysore
1802-4	Rajputana, Bombay, one of the worst on record	1868-70	Parts of Northwestern Provinces, Gujarat, Punjab, Deccan, parts of Central Provinces, terrible mortality in Rajputana
1806-7	Widespread	1873-74	Behar, Bundelkhand, Southwestern Provinces, "Panic Famine"
1812-13	Bombay, Agra, and Madras districts	1876-78	Madras, Bombay, Mysore, Hyderabad, an estimated 5 million dead
1819-20	Northwestern Provinces, Rajputana, Deccan, Broach, panic, migration	1877-78	United Provinces, Kashmir, Ganjam, Native Orissa States
1820	Upper Sind	1896-97	Widespread
1822	Upper Sind	1899-1900	Widespread, perhaps the greatest famine on record
1824-25	Deccan, Bombay, Madras, parts of Northwestern Provinces	1900-2	Gujarat
1832-33	Sholapur, northern Madras	1906-7	Darbhanga district, floods
1833-34	Gujarat, Kandish, northern Deccan	1907	United Provinces
1837	Northwestern India, 2 years		

References, Abbreviations, and Indexes

References

THE references cited throughout Volumes I and II are given here. In all but a few rare cases the originals have been consulted. The abbreviations used for journal names are listed below; for these we have in general omitted all articles, prepositions, and conjunctions. The volume numbers, followed by a comma, precede the inclusive page numbers; thus Vol. 7, pages 15 through 36, is written 7, 15-36.

Abstr., Abstracts	Dent., Dentistry, Dental
Acad., Academy, Académie, Académique	Dermat., Dermatology, Dermatologie, etc.
Adv., Advances, Advancement	Deut., Deutsch(es)
Agric., Agriculture, Agricultural	Digest., Digestive
Akad., Akademie, Akademisch	Dis., Disease(s)
Am., America, American	Diss., Dissertation
An., Anales, Anais	Ed., Editor
Anat., Anatomy, Anatomie, Anatomia, Anatomical, etc.	Endocrinol., Endocrinology
Ann., Annals, Annales, Annali, Annual	Ergebn., Ergebnisse
Anthropol., Anthropology	Españ., Español(a)
Anz., Anzeiger	Exp., Experimental, Experimentell(e), Expérimental
Arch., Archives, Archiv, Archivio, Archivo(s)	Fac., Faculty, Facultad
Arq., Arquivos	Fed., Federation
Asoc., Asociación	Franç., Français(e)
Assoc., Association	Gac., Gaceta
Austral., Australian	Gaz., Gazette, Gazeta
Bact., Bacteriology, Bacteriologie	Gazz., Gazzeta
Bakt., Bakteriologie	Geburtsh., Geburtshilfe
Beitr., Beiträge	Gen., General
Belg., Belgisch, Belge, Belgique	Geneesk., Geneeskunde
Ber., Berichte	Gerontol., Gerontology, Gerontological
Berl., Berlin or Berliner	Ges., Gesamte
Bioch., Biochemistry, Biochemical, Bio- chemie, Biochimie, etc.	Gesell., Gesellschaft
Biol., Biology, Biologie, Biological, etc.	Gior., Giornale
Bol., Boletin(es)	Grad., Graduate
Boll., Bollettino	Gynäk., Gynäkologie
Brit., Britain, British	Gynec., Gynecology, Gynecologie
Bull., Bulletin(s)	Heilk., Heilkunde
Canad., Canadian, Canadienne	Helv., Helvetica
Cbl., Centralblatt	H.M., His Majesty's
Chem., Chemistry, Chemical, Chemie, etc.	Hôp., Hôpital
Child., Children, Childhood	Hosp., Hospital
Chir., Chirurg, Chirurgie, Chirurgia, Chirurgiae, etc.	Hyg., Hygiene, Hygienic
Cir., Cirugia, Cirujano	Immunol., Immunology
Clin., Clinic, Clinical, Clinica	Indust., Industrial
Comp., Comparative	Inform., Informaciones
Cong., Congress, Congrès, Congreso	Inn., Inner(e)
C. r., Comptes rendus	Inst., Institute, Institut, Institution
	Internat., International(es)
	Invest., Investigation
	Ital., Italiano

- J., Journal
 Jber., Jahresberichte
 Jg., Jahrgang
 Kinderh., Kinderheilkunde
 Klin., Klinik, Klinisch
 Krankh., Krankheit
 Lab., Laboratory, Laboratoire, etc.
 Lat., Latino
 Med., Medicine, Médecin, Medizin, Medica,
 etc.
 Mem., Memoires, Memories, Memorias
 Metab., Metabolic
 Mex., Mexico, Mexicano
 Mil., Military
 Mitt., Mitteilungen
 Mo., Monthly
 Monats., Monatschrift
 Nat., National(es)
 Nederl., Nederlandsch
 Néerl., Néerlandais
 Neurol., Neurology, Neurologie, etc.
 Occup., Occupational
 Path., Pathology, Pathologie, etc.
 Ped., Pediatrics, Pediatrica
 Pharm., Pharmacy
 Pharmaceut., Pharmaceutical
 Pharmacol., Pharmacology, Pharmacologie
 Pharmakol., Pharmakologie
 Phys., Physics, Physik, Physical, etc.
 Physiol., Physiology, Physiologie, etc.
 Proc., Proceedings
 Psychiat., Psychiatry, Psychiatric
 Psychol., Psychology, Psychological
 Publ., Public, Publication
 Quart., Quarterly
 Rad., Radiology, Radium, etc.
 Rec., Record
 Res., Research
 Rev., Review(s), Revista, Revue
 Riv., Rivista
 Roy., Royal
 Scand., Scandinavia, Scandinavica, etc.
 Schweiz., Schweizerisch
 Sci., Science, Scientific
 Sect., Section
 Sem., Semaine
 Skand., Skandinavisch
 Soc., Society, Société, etc.
 Sociol., Sociology, Sociological
 Sovet., Sovetskaya
 Suppl., Supplement, Supplemental,
 Supplementum, etc.
 Surg., Surgery
 Syph., Syphilology
 Therap., Therapy, Therapeutic
 Tijds., Tijdschrift
 Tr., Transactions
 Trop., Tropical
 Tuberc., Tuberculosis
 Ugesk., Ugeskrift
 Univ., University, Université, Universidad
 Ws., Wochenschrift
 Z., Zeitschrift
 Zbl., Zentralblatt
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Abbreviations, Minnesota Experiment

All the abbreviations used in the text have been defined in the text proper or in the Appendix. To facilitate the reading of single chapters, the abbreviations needed for the understanding of the text and particularly of the tables are summarized here.

Periods

C = control (12 weeks), devoted to standardization and training in the test procedures.

S = semi-starvation (24 weeks).

R = controlled rehabilitation (12 weeks).

The number following the period symbol indicates the approximate week of the period during which the observations were made (e.g., S24). The control values were obtained during C10 to C12.

Dietary Groups during the First 12 Weeks of the Rehabilitation Period

Z = "+0" = "Zero" caloric group; mean actual intake from R1 to R12, 2378 Cal.

L = "+400" = "Low" caloric group; 2692 Cal.

G = "+800" = "Good" caloric group; 3123 Cal.

T = "+1200" = "Top" caloric group; 3392 Cal.

U = "Unsupplemented" with protein.

Y = "Yes," supplemented with protein.

P = "Placebo," unsupplemented with vitamins.

H = "Hexavitamin," supplemented with vitamins.

Statistical Symbols

$d = S - C$, the difference between the mean values obtained during the semi-starvation and the control period. Most frequently, $d = S24 - C$, the difference between values obtained at the end of the semi-starvation period and the control values. In some instances the symbol d was used for the mean difference between other periods; this is noted specifically in each case.

$\Delta = R - S24$, the difference between the mean rehabilitation and final semi-starvation measurements. Most frequently $\Delta = R12 - S24$.

V_{bGr} = "between-group" variance.

V_{rep} = "replicate" (error) variance.

F = a statistical test of the significance of the difference between 2 or more means. It is always a ratio of 2 variances; in the rehabilitation period $F = V_{bGr}/V_{rep}$ unless specified otherwise.

[*] = the F ratio reached or exceeded the 5 per cent level of statistical significance ($F_{0.05}$).

[**] = the F ratio reached or exceeded the 1 per cent level of statistical significance ($F_{0.01}$).

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